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A rare case of complex ventricular arrhythmia and heart failure in a 15.5-year-old athlete

Rzadki przypadek złożonej arytmii komorowej i niewydolności serca u 15,5-letniego sportowca

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Abstract

A 15.5-year-old boy, an athlete, reported to his general practitioner due to episodes of chest pain during exercise, weakness and a feeling of irregular heartbeat that had been occurring for a month. He reported episodes of fainting in the past. Physical examination revealed arrhythmia and the patient was referred for a cardiological consultation, and then admitted to a reference cardiology department for extended cardiac diagnosis. Approximately 66,000/day (49.8%) of premature ventricular beats were recorded in 24-hour Holter electrocardiogram, including episodes of non-sustained ventricular tachycardia. Echocardiography revealed left ventricular systolic dysfunction with a decrease in its ejection fraction to 51.5%. Magnetic resonance imaging of the heart was performed, which showed foci of late post-contrast enhancement, located subepicardialy in the side wall of the left ventricle, corresponding to post-inflammatory changes in the myocardium. Left and right ventricular ejection fraction on magnetic resonance imaging were 48% and 46%, respectively. Pharmacological treatment included a beta-blocker (metoprolol), propafenone and an angiotensin converting enzyme inhibitor (enalapril). During the treatment, ventricular arrhythmia subsided almost completely, a gradual improvement in left ventricular systolic function was observed, and the patient was relieved of pain. The boy was discharged home with a recommendation to continue pharmacological treatment, lead a sparing lifestyle, and postpone practicing sports. At present, the boy's general condition is good, he does not report any complaints, takes medications regularly, and does sports only for recreation. A follow-up Holter showed only single premature ventricular beats, while echocardiography showed an improved left ventricular systolic function (ejection fraction 63%), which is a good prognostic indicator.

Keywords: sports, ventricular arrhythmia, myocarditis, heart failure

Streszczenie

Chłopiec 15,5-letni, uprawiający sport, zgłosił się do lekarza rodzinnego z powodu występujących od miesiąca epizodów bólu w klatce piersiowej podczas wysiłku, osłabienia oraz uczucia niemiarowej czynności serca. Poinformował też o epizodach zasłabnięcia w przeszłości. W badaniu przedmiotowym stwierdzono arytmię i skierowano pacjenta na konsultację kardiologiczną, a następnie przyjęto do referencyjnego oddziału kardiologicznego w celu rozszerzenia diagnostyki. W 24-godzinnym badaniu elektrokardiograficznym metodą Holtera zarejestrowano około 66 000 dodatkowych pobudzeń komorowych na dobę (49,8%), w tym epizody nieutrwalonego częstoskurczu komorowego. W badaniu echokardiograficznym stwierdzono upośledzenie czynności skurczowej lewej komory, z obniżeniem jej frakcji wyrzutowej do 51,5%. Wykonano badanie serca metodą rezonansu magnetycznego, w którym uwidoczniono ogniska późnego wzmocnienia pokontrastowego, zlokalizowane podnasierdziowo w ścianie bocznej lewej komory, odpowiadające przebytym zmianom zapalnym w mięśniu sercowym. Frakcja wyrzutowa lewej komory w rezonansie magnetycznym wynosiła 48%, prawej – 46%. W leczeniu farmakologicznym zastosowano beta-adrenolityk (metoprolol), propafenon oraz inhibitor enzymu konwertującego angiotensynę (enalapril). W trakcie terapii uzyskano niemal całkowite ustąpienie arytmii komorowej, jak również stopniową

poprawę funkcji skurczowej lewej komory, ponadto u pacjenta ustąpiły dolegliwości bólowe. Z zaleceniem kontynuacji leczenia farmakologicznego oraz prowadzenia oszczędzającego trybu życia i odroczenia uprawiania sportu pacjent został wypisany do domu. Obecnie jego stan ogólny jest dobry, chłopiec nie zgłasza dolegliwości, regularnie przyjmuje leki, sport uprawia jedynie w zakresie rekreacyjnym. W kontrolnym badaniu elektrokardiograficznym metodą Holtera zarejestrowano tylko pojedyncze dodatkowe pobudzenia komorowe, natomiast w badaniu echokardiograficznym stwierdzono poprawę funkcji skurczowej lewej komory (frakcja wyrzutowa 63%), co jest dobrym wskaźnikiem prognostycznym.

Słowa kluczowe: sport, arytmia komorowa, zapalenie mięśnia sercowego, niewydolność serca

INTRODUCTION

Practicing sports is an attractive and recommended form of spending time, especially now when working with tablets, smartphones and other screen-based electronic devices, regardless of the purpose for which these are used, often consumes a significant part of the day. Arrhythmias can affect cardiac haemodynamic parameters and decrease the left ventricular ejection fraction (LVEF), which significantly compromises exercise capacity. The guidelines of the European Society of Cardiology (ESC) indicate that ventricular arrhythmias occur in the majority of patients with heart failure (HF) and that sudden deaths are common in this population[1,2]. Prevention of sudden cardiac death (SCD) in athletes includes recommendations for a detailed medical and family history (class I, level C) for cardiovascular pathology, arrhythmias and episodes of syncope. Sudden cardiac death is the leading cause of exercise-related mortality in athletes[3–5]. SCD in young athletes is caused by structural and electrophysiological myocardial disorders, including cardiomyopathies, channelopathies, anomalies of the coronary arteries, as well as congenital heart defects and acquired myocardial pathologies[3,6–11]. The incidence of SCD among competitive athletes is currently estimated to range from 1/5,000 to 1/1,000,000 per year[2,3]. According to Harmon et al., a study conducted among American students found that men were at a higher risk of SCD than women (1/38,000 vs. 1/122,000), while black athletes had a 3.2-fold higher risk of SCD than in white athletes (1/21,000 vs. 1/68,000). According to this research, black male basketball players (US) and footballers (Europe) are among the highest risk groups for sports-related SCD[3]. We present a case of a 15.5-year-old male athlete diagnosed with complex ventricular arrhythmia and HF symptoms associated with a reduced right ventricular ejection fraction (RVEF), possibly of post-inflammatory aetiology. Pharmacological treatment and exercise restriction were found to be an effective therapeutic approach (the number of premature ventricular beats was significantly reduced). If ventricular arrhythmia occurs in the course of genetically determined diseases, e.g. long QT syndrome (LQTS) or Brugada disease[7,12], treatment is very difficult and the prognosis is uncertain. In such cases, qualification for resuming competitive sports should be made by a sports medicine practitioner.

CASE REPORT

A 15.5-year-old boy, physically well developed (body weight 63 kg, height 177 cm, body mass index 20.1 kg/m^2), reported to his general practitioner (GP) due to episodes of chest pain during exercise, weakness and a feeling of irregular heartbeat. He also reported recurring exercise-unrelated episodes of syncope at rest in the past. He attended a uniformed services school, was a very active amateur athlete, cycled up to 100 km a day, exercised in the gym and practiced bodybuilding. He denied the use of anabolic steroids and stimulants. Physical examination performed by his GP found an irregular heartbeat and, after a clinical examination, electrocardiography (ECG) was ordered, in which single premature ventricular beats were recorded. For this reason, the patient was referred to the department of cardiology. At the time of consultation, he was cardiovascularly stable and had normal vital signs, including blood pressure. The heart rate was irregular, with multiple audible additional contractions. The peripheral pulse was symmetrical and strong. Echocardiography showed no congenital heart defect, but attention was drawn to the impaired systolic function and decreased LVEF calculated using the Teichholz method (51.5%; normal: ≥55%) (Fig. 1). The patient was referred for urgent admission to the department of cardiology. A 24-hour Holter showed complex ventricular arrhythmia with single premature beats (PBs), pairs of PBs and episodes of non-sustained ventricular tachycardia, a total of approximately 66,000 (49.8%) premature ventricular beats per day (Fig. 2). The treatment was started with a beta-blocker (metoprolol) at a dose of 50 mg in the morning and 25 mg in the evening. Due to the lack of therapeutic efficacy, propafenone was included at a dose of 3 × 150 mg. Follow-up Holter showed significantly reduced number of premature ventricular beats and reduced severity of ventricular arrhythmia, while echocardiography still showed reduced LVEF calculated by the Teichholz method (50%; normal: ≥55%). The treatment was supplemented with an angiotensin converting enzyme inhibitor (enalapril) at a dose of 2 × 5 mg. During the exercise test, ventricular arrhythmia exacerbated in its initial stage, after which it was completely "extinguished" and resolved during the fourth stage of the test (peak exercise), to recur in the first minute after the end of the test. Due to the nature of the diagnosed ventricular arrhythmia as well as impaired systolic function and reduced LVEF, magnetic resonance imaging

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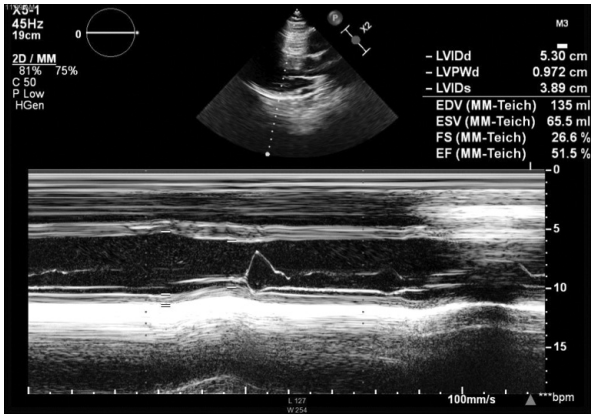

Fig. 1. M-mode echocardiography. Parasternal line in the long axis. Reduced LVEF calculated by the Teichholz method (51.5%)

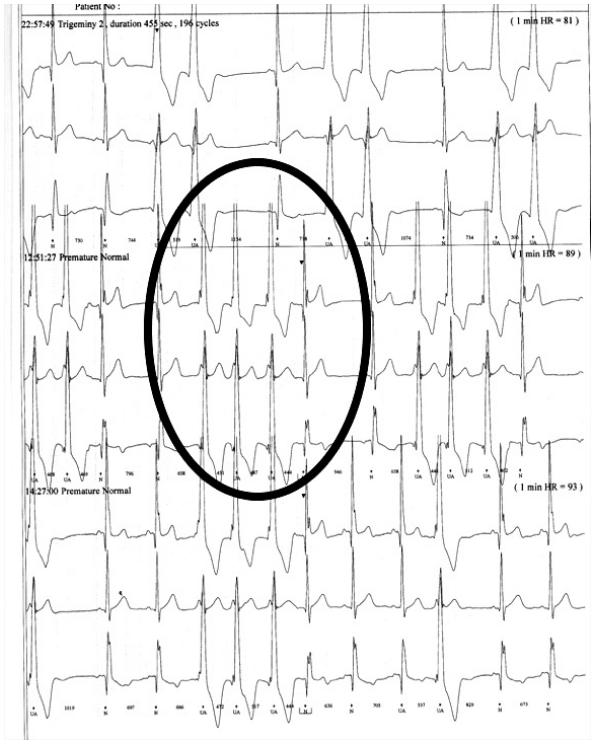

Fig. 2. Electrocardiogram – 24-hour Holter recording on admission. Single premature ventricular beats, pairs, and an episode of non-sustained ventricular tachycardia (three consecutive premature ventricular beats – circled)

(MRI) of the heart was performed, which revealed pathological subepicardial foci of enhancement within the lateral walls of the left ventricle, at the level of the middle segments (late gadolinium enhancement MRI, LGE-MRI) (Fig. 3). Post-inflammatory changes were identified within the left ventricular lateral wall, with a significantly reduced left ventricular ejection fraction (LVEF = 48%), and in the right ventricle (RVEF = 46%). During hospital stay at the department of cardiology, the patient was stable and did not report any complaints. The pharmacological treatment used turned out to be effective. A follow-up 24-hour

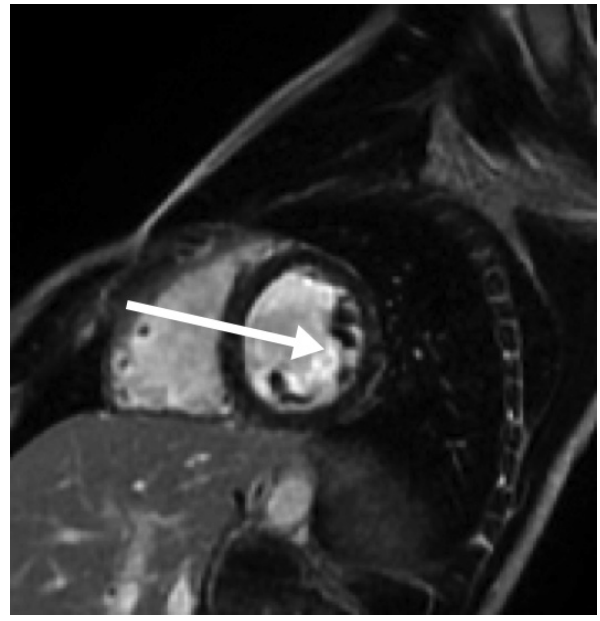

Fig. 3. Magnetic resonance imaging of the heart. Reduced LVEF and RVEF. The images of late enhancement after contrast administration show spotted areas of pathological enhancement of the myocardium in the subepicardium in the lateral wall of the left ventricle, at the level of the middle segments. Post-inflammatory lesions were identified within the left ventricular lateral wall, with a significantly reduced left ventricular ejection fraction (LVEF = 48%) and in the right ventricle (RVEF = 46%)

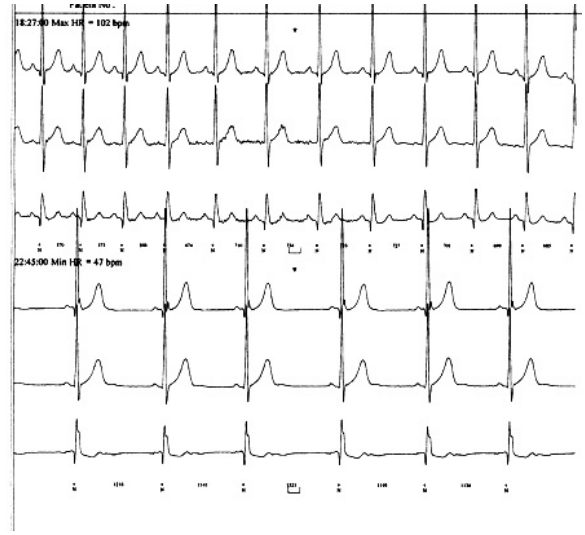

Fig. 4. Electrocardiogram – 24-hour Holter recording on discharge. Only 20 single premature ventricular beats per day were recorded (not included on the electrocardiogram)

Holter recorded only 20 single premature ventricular beats (Fig. 4). The boy complied with medical and nursing recommendations and was discharged home with instructions to continue pharmacological treatment, limit physical exercise to recreational level, as well as report for follow-up tests at the department of cardiology in 4 weeks, and a follow-up MRI scan in 6 months.

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DISCUSSION

The presented case report of a patient practicing sports illustrates the initial diagnosis properly performed by a GP and his correct decisions, as well as the subsequent stages of the diagnostic and therapeutic process. The seemingly innocent arrhythmia, which is often diagnosed in young patients, had several unusual aspects in this patient that are worth discussing. Despite the patient's reluctance to provide information about his health (the boy tried to dissimulate the disease), the symptoms reported by his caregivers could not be underestimated, as rapid diagnosis and implementation of treatment probably prevented the serious and even life-threatening consequences of untreated cardiac arrhythmia in this case[1–3,5–7,12]. The coexistence of intensive sport practice, ventricular arrhythmia, greater than usual weakness and syncope episodes are an alarm signal in a young patient. In the described patient, symptoms of heart failure related to impaired systolic function and decreased LVEF were also present. Ventricular arrhythmia subsided during the exercise stress test to recur after the end of the test, when the patient was at rest. Athletes who deny problems often say: "physical exercise is not harmful to me because then I feel great." It is true that during exercise they do not feel any discomfort, but they usually do not mention that when at rest rhythm disturbances quickly return and often intensify even more – as in the described patient. Young people often associate their future with practicing professional sports, but – as shown in this paper – there are health situations that make it impossible to do so in individually varied periods of time. Currently, in the era of the coronavirus disease 2019 (COVID-19) pandemic, patients report, among others, episodes of palpitations, general weakness, chest pain, reluctance to exercise, apathy and even symptoms of beginning or worsening depression[13]. No signs of acute viral infection were found in our patient and the PCR test for coronavirus infection was negative. In addition, the boy was vaccinated twice against the coronavirus infection. Particularly noteworthy is the fact that the patient had a severe complex ventricular arrhythmia, as young people rarely present with up to 50% of premature ventricular beats, including episodes of non-sustained ventricular tachycardia, with a simultaneous feeling of full health[14]. Cardiac MRI findings indicated a history of myocarditis, but it was difficult to specify the actual onset of inflammation. Other patients hospitalised at the Department of Paediatric Cardiology of the Upper Silesian Child Health Centre in Katowice due to acute viral myocarditis presented mainly with symptoms of acute infection, acute chest pain, high C-reactive protein (CRP), leukopenia (less commonly leukocytosis), significantly elevated troponins (several hundred or even several thousand, with the norm up to 14 ng/L) and repolarisation abnormalities in resting ECG. Echocardiography showed a decreased LVEF and impaired myocardial contractility only in some patients. In previously hospitalised patients, LVEF was most often normal or in "the lower limit of normal." There were no signs of viral infection in the described patient, troponin levels were normal, and no changes in the ST segment or T-wave were found in ECG or Holter ECG[14]. Thus, there was no basis for the diagnosis of an acute inflammatory myocardial process, the more that virological tests were negative. On the other hand, a very disturbing symptom was a decreased LVEF (48%), which improved, but the boy requires further cardiological follow-up. Antiarrhythmic treatment was found to be highly effective, but the fact that the boy did not perform any physical exertion during hospital stay should also be taken into account. Therefore, perhaps these were rest, the pharmacotherapy used and myocardial regeneration during the patient's hospital stay that essentially improved the systolic function of the heart and reduced the severity of ventricular arrhythmia, especially since MRI revealed "past' rather than "ongoing" myocarditis. The episodes of syncope reported before hospitalisation were an additional disturbing symptom, and although they were not shown to be related to exercise, fainting in patients with ventricular arrhythmia should always be a reason for concern, as they may be a symptom of a ventricular tachycardia, low cardiac output syndrome and a risk factor for SCD[3–7]. During hospitalisation, the described patient experienced one episode of syncope during blood collection, but its course suggested a reflex nature.

CONCLUSIONS

Ventricular arrhythmia coexisting with heart failure in an active athlete can pose a real health and life threat. Early diagnosis and appropriate treatment can prevent the dangerous consequences of the disease.

Conflict of interest

The authors do not declare any financial or personal links to other persons or organisations that could adversely affect the content of this publication or claim rights thereto.

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