



MALIGNANT RHYTHM DISORDER OCCURRING IN AN ASYMPTOMATIC CHILD DURING PHYSICAL ACTIVITY

KANITA KAMENICA¹, SENKA MESIHOVIĆ-DINAREVIĆ²

Heart-rhythm disorders and related congenital anomalies in children often remain silent until physical activity brings on symptoms. We report an 11-year-old girl who experienced her first signs of heart disease only after exertion. Although late detection of such anomalies can sometimes be fatal, careful clinical examination may reveal these conditions early.

Keywords: MALIGNANT RHYTHM, PHYSICAL ACTIVITY, ASYMPTOMATIC CHILD, LEFT AXIS DEVIATION

INTRODUCTION

Malignant rhythm disorders in asymptomatic children, particularly when they occur during physical activity, represent a critical concern in paediatric cardiology. Although rare, sudden cardiac arrest (SCA) in children often occurs in those who appear healthy but carry underlying electrical or structural cardiac abnormalities. The principal underlying factors include inherited channelopathies (such as Long QT Syndrome, Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) and cardiomyopathies (Hypertrophic Cardiomyopathy (HCM), Arrhythmogenic Cardiomyopathy (ACM) (1). During exercise or intense activity, the boost in catecholamines and increased hemodynamic stress can unmask electrical instability, precipitating malignant ventricular arrhythmias, syncope, or even sudden death (SD) (2).

Myocardial scars caused by pre-existing cardiac anomalies that were not detected represent an irreversible substrate

for malignant arrhythmias, which can be induced by physical activity, making the consideration of an Implantable Cardioverter-Defibrillator (ICD) for secondary prevention advisable (3).

CASE REPORT

An 11-year-old girl was admitted to the Intensive Care Unit (ICU), intubated and ventilated with a Glasgow Coma Scale (GCS) of 6, after suffering cardiac arrest during training. On-site Cardiopulmonary resuscitation (CPR) restored circulation, and the electrocardiogram (ECG) showed ventricular fibrillation (VF) which was defibrillated to a sinus rhythm. Brain Magnetic Resonance Imaging (MRI) was normal. She remained on mechanical ventilation for 48 hours and was extubated on day 3, after which she was transferred to the cardiology unit in a stable condition. Prior to the event, she was healthy, actively training, with occasional headaches. Family history revealed the father's atrial fibrillation, prompting genetic testing: a pathogenic mutation of the TMEM43 gene and a variant in the CACNA1H gene were found, suggesting increased genetic risk. Echocardiography revealed no significant heart chamber enlargement, a dilated right coronary artery and improper visualisation of the left coronary origin. Computed Tomography (CT) coronary angiography confirmed an anomalous

left coronary artery from the pulmonary artery (ALCAPA), and surgical correction was recommended. She was treated with diuretics, propranolol, and phenobarbitone. Pre-arrest sports check-up had shown a systolic murmur I-II/V₁, normal pulses, an ECG shows: a normal sinus heart rhythm with 60 bpm, LAD, PR int 130 ms, QT int 320 ms, in aVR dominant rSR' wave, aVF dominant S wave up to 9mm, IRBBB in V₁ and V₂, R wave in V₆, no q waves, normal ST segment (Figure 1); Holter: 99% regular QRS, mean 91 bpm, rare Ventricular Extrasystoles (VES) (0.03%); Transthoracic Echocardiogram (TTE): LVEF 37%. Two months later, she underwent successful surgical transfer of the left coronary artery to the aorta and has since done well, gaining 9 kg, no complaints, on bisoprolol. At four months post-surgery: sat 96%, HR 71 bpm, ECG: sinus rhythm 65 bpm, LAD, PQ 140 ms, QT 380 ms; echocardiography: normal atria/ventricles, left ventricular ejection fraction (LV EF) 64% in 3D volumetry. Cardiac MRI perfusion showed neither fibrosis nor scar and no criteria for arrhythmogenic cardiomyopathy. She continues on a beta blocker and avoids heavy exertion until a stress test in three months. Ongoing surveillance is essential because myocardial scarring constitutes an irreversible substrate for malignant ventricular arrhythmias and recurrent events.

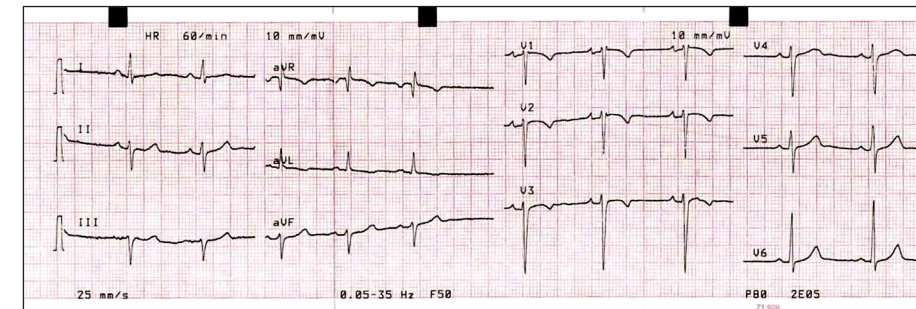


Figure 1.
12-lead ECG/see earlier description in the text/

DISCUSSION

The normal heart rhythm is the sinus rhythm, which means it is initiated by electrical impulses originating from the sinoatrial node (SAN) - the heart's natural pacemaker. In a normal sinus rhythm, the P wave measures under 120 ms in duration and ≤ 0.25 mV in amplitude, with a consistent P-P interval on the ECG (4). Physiologically, the cardiac axis, which represents the direction of electrical impulse conduction through the heart, normally ranges from -30° to $+90^\circ$ (5). Our patient had a sinus rhythm but a left axis deviation (LAD), with the electrical axis shifted between -30° and -90° . In children, the cardiac axis is typically within the normal range; however, deviations can occur and are often associated with structural heart diseases. LAD may indicate an underlying cardiac condition, but can also be seen in healthy children (6).

Physical activity (PA) is defined as any bodily movement produced by skeletal muscles that results in energy expenditure, and it is essential for long term cardiovascular health in patients with Chronic Heart Disease (CHD) (7). The American Heart Association recommends that children with CHD have at least 60 minutes of moderate-to-intense physical activity every day, with limitations for those with arrhythmias or significant left ventricular outflow tract problems. In a study conducted by Voss C and colleagues in 2017, the cardiologist-prescribed physical activity restrictions were uncommon ($\approx 15\%$) and generally limited to intense isometric exercises, mainly for patients with left ventricular outflow tract abnormalities, such as a bicuspid aortic valve or aortic coarctation (8). Christian et al. found that around

half of paediatric patients with channelopathies and cardiomyopathies reduced physical activity and reported lower quality of life after diagnosis. Exercise restrictions in these children may increase obesity risk and contribute to long-term cardiovascular deconditioning (9).

Arrhythmogenic cardiomyopathy (ACM) is a genetic heart muscle disease caused by defects in cell-to-cell adhesion structures, mainly desmosomes. The condition is characterized by gradual myocyte loss and replacement with fibrofatty tissue (scars), which predisposes to re-entrant ventricular tachycardia (VT) and cardiac dysfunction (10). Physical activity (sport) is a recognized trigger for arrhythmias, and ACM is a major cause of sudden death (SD) in athletes, both male and female (11, 12). In the review by A. Zorzi et al. in 2021, they highlight the importance of systematic preparticipation screening, because it lowers the risk of sudden death in classic ACM forms (right-dominant or biventricular) by enabling early detection of asymptomatic patients through abnormal ECG findings or exercise-induced ventricular arrhythmias (10).

Myocarditis is a significant but frequently overlooked cause of sudden cardiac death in young people and athletes. Although many individuals experience prior symptoms such as viral illness, palpitations, chest pain, or syncope - the condition often remains undiagnosed before death. Involvement of the heart's conduction system can trigger arrhythmias, making myocarditis a hidden risk even when standard cardiac evaluations are normal (13).

Genetic testing is a key aspect of personalized medicine, identifying disease-causing variants and is best suited for patients with suspected or confirmed inherited cardiovascular disease or a known familial pathogenic variant (13, 14). It identifies pathogenic variants in about 10% of apparent Unexpected cardiac arrest (UCA) survivors, primarily in cardiomyopathy-related genes, indicating their arrhythmogenic role even without clear cardiomyopathy. These results support genetic testing of arrhythmia and cardiomyopathy genes in UCA survivors (15). In our case, genetic testing revealed variants in the TMEM43 and CACNA1H genes, suggesting a combined genetic burden. These mutations are associated with cardiovascular disorders and, in this instance, may have contributed to the development of malignant arrhythmias, including AF and ALCAPA. Also a positive family history, as well as the presence of symptoms in individuals engaged in sports, will require further reevaluation, because the absence of a detected arrhythmia during initial diagnostic testing does not necessarily mean that one is not present (17).

Considering that cardiac events can occur sporadically and without warning, using a smartwatch with ECG capability can serve as a valuable additional "safety-net" tool. Modern smartwatches equipped with ECG apps allow users to quickly record their heart's electrical activity at the onset of symptoms such as during exercise, physical strain, dizziness, palpitations, or other discomfort providing supplementary information to that obtained through standard clinical evaluations (17, 18).

In the end shared decision-making is essential for athletes with cardiovascular conditions, allowing personalized, informed choices about participation in sports. It improves understanding of risks, aligns management with the athlete's values, and considers physical, psychological, and social factors, while helping to responsibly manage potential cardiovascular risks (19).

¹Javna ustanova Dom zdravlja Kantona Sarajevo
²Eurofarm Poliklinic, Sarajevo

Corresponding author:
Dr. Kanita Kamenica
Javna ustanova Dom zdravlja Kantona Sarajevo
71000 Sarajevo, Bosnia and Herzegovina,
Nerkesijina 17
E-mail: kamenikanita@gmail.com

CONCLUSION

While children may present as fit and healthy, underlying malignant rhythm disorders can lead to life threatening events during physical activity. Athletes should first be assessed by a paediatric cardiologist or, if needed, by a genetic specialist. Those with symptoms should avoid competitive sports until properly informed on treatment and symptom-free for three months. Recognizing high risk features and appropriate screening and managing is vital to prevent tragic outcomes. In adolescents and young adults, physical activity can trigger SD in those with underlying heart conditions, though exercise itself does not increase overall mortality and remains beneficial for cardiovascular health. In individuals in whom a cardiac anomaly is detected incidentally on clinical examination, personal history of family members, echocardiography or ECG shows specific characteristics, monitoring is required. Particularly if MRI of myocardium reveals the presence of fibrotic scars that may serve as a source of malignant arrhythmias - in our case, ventricular fibrillation - the implantation of an ICD for secondary prevention should be considered due to possible recurrent cardiac arrests and VF. For athletes - especially those with cardiac condition, the practical use of ECG-enabled smartwatches is a highly valuable tool. They allow the recording of abnormal heart rhythms during physical activity or other symptomatic episodes and can serve as a complementary tool alongside standard diagnostic evaluations.

Abbreviations:

SCA - Sudden cardiac arrest
CPVT - Catecholaminergic Polymorphic Ventricular Tachycardia
ACM - Arrhythmogenic Cardiomyopathy
SD - Sudden death
ICD - Implantable Cardioverter-Defibrillator
ICU - Intensive Care Unit
GSC - Glasgow Coma Scale
CPR - Cardiopulmonary Resuscitation
ECG - Electrocardiogram
VF - Ventricular Fibrillation
MRI - Magnetic Resonance Imaging
CT - Computed Tomography
ALCAPA - Anomalous Left Coronary Artery from the Pulmonary Artery
BPM - Beats Per Minute
VES - Ventricular Extrasystoles
TTE - Transthoracic Echocardiogram

LV EF - Left Ventricular Ejection Fraction
SAN - Sinoatrial Node
CHD - Congenital Heart Disease
UCA - Unexpected cardiac arrest

NOVČANA POTPORA/*FUNDING*
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Sažetak

MALIGNI POREMEĆAJ SRČANOG RITMA KOD DIJETETA BEZ SIMPTOMA ZA VRIJEME TJELESNE AKTIVNOSTI

Kanita Kamenica, Senka Mesihović-Dinarević

Poremećaji srčanog ritma i udružene urođene srčane anomalije mogu se otkriti kod djece. U nekim slučajevima tjelesna aktivnost može izazvati pojavu simptoma koji do tada kod djeteta nisu bili prisutni. Prikazujemo slučaj jedanaestogodišnje djevojčice koja nije imala nikakve simptome srčanog oboljenja sve dok fizička aktivnost nije pokrenula njihovu pojavu. Kasno otkrivanje srčanih anomalija ponekad može biti pogubno za pojedinca - a iste se mogu otkriti detaljnim kliničkim pregledom.

Ključne riječi: MALIGNI RITAM, FIZIČKA AKTIVNOST, ASIMPTOMATSKO DIJETE, DEVIJACIJA LIJEVE ELEKTRIČNE OSOVINE

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