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Ventricular fibrillation leading to sudden cardiac arrest in an asymptomatic child with left axis deviation and anomalous left coronary artery from the pulmonary artery

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ABSTRACT

Anomalous left coronary artery from the pulmonary artery (ALCAPA) is a rare heart defect present at birth. In this condition, the left coronary artery, which normally comes from the aorta, instead starts from the pulmonary artery. This abnormal connection can lead to reduced blood flow to the heart muscle, causing damage like heart attacks and increasing the risk of serious heart rhythm problems or sudden cardiac arrest (SCA). Diagnosis is made through imaging studies, and treatment often involves surgical correction to restore normal coronary circulation. The prognosis of ALCAPA largely depends on the age at which it is diagnosed and the timing of treatment. Early intervention is crucial for improving outcomes. This case report presents a 11-year-old girl, who is an athlete, with late presentation of ALCAPA syndrome in the form of ventricular fibrillation leading to SCA. She had no previous symptoms, the electrocardiogram showed no abnormalities except for left axis deviation and the several echocardiograms showed no enlargement of the left ventricle, with possible big right coronary artery. ALCAPA has been confirmed by computed tomography coronary angiography scan. Child was surgically treated by coronary artery transfer for ALCAPA with satisfactory outcome. The implantation of an implantable cardioverter defibrillator is still under consideration.

Keywords: Ventricular fibrillation; sudden cardiac arrest; left axis deviation; anomalous left coronary artery from the pulmonary artery

INTRODUCTION

Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA), also known as Bland-White-Garland Syndrome, is a congenital cardiac anomaly that, while uncommon, poses significant health risks. It may result in fatal outcomes during infancy and, in certain instances, cause sudden death in adulthood (1). This condition is divided into two forms: infant and adult form, depending on whether or not alternative blood vessels (coronary collaterals) have formed to help supply the heart. Even with compensatory mechanisms in adult patients, the risk of sudden death remains high, with an estimated incidence of 80 to 90% around the average age of 35 years (2). If left untreated, the mortality rate in the first year of life is a 90% due to heart failure, ischemic damage to the heart muscle and sudden cardiac death (SCD) (3). However, if diagnosed early and surgically corrected, the prognosis can be favourable, with many patients experiencing a near-normal life expectancy and quality of life after surgery.

The heart's electrical axis represents the overall direction of electrical impulses as they move through the heart. This axis is quantified in degrees on the frontal plane, with a normal range spanning from −30° to +90°. Left axis deviation (LAD) occurs when the axis shifts leftward, falling between −30° and −90° (4). Even though it is rare in children, may be associated with structural heart disease, but may also be observed in healthy children. The severity of LAD is linked to the chance of having congenital heart disease (CHD). A physical exam plays an important role in detecting CHD in people who show signs of LAD (5,6). As a result, this condition seems harmless in the short term, but longer follow-up is needed to fully understand the long-term outcome. LAD is considered a borderline finding in athletes, but when it appears alongside another borderline condition, like a right bundle branch block, it warrants further evaluation due to the potential increased risk of SCD.

Physical activity (PA) in children with CHD is essential for improving long-term health. In CHD populations, regular PA has been shown to improve heart function or help

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slow the worsening of conditions like systolic and diastolic heart failure, pulmonary hypertension, and coronary artery disease. Exercise also boosts muscle strength and aerobic fitness, reduces the risk and occurrence of heart and metabolic conditions, promotes healthier habits, and supports a more active lifestyle (7). Exercise limitations were considered to have a minor influence on the chances of dying from any cause (8).

This case report highlights an 11-year-old athletic girl who was diagnosed with ALCAPA syndrome later than usual, after suffering VF that led to cardiac arrest.She had no previous symptoms, the electrocardiogram (ECG) showed no abnormalities, no abnormal q waves in I, aVL, V5, V6, no ST changes except for LAD, the transthoracic echocardiogram (TTE) was normal with no enlargement of the left ventricle, except big right coronary artery. Child was operated via coronary artery transfer for ALCAPA with satisfactory outcome. The ICD implantation due to possible recurrent cardiac arrests and VF is still under consideration, having in mind her genetic substrate/variant of unclear significance in the transmembrane protein 43 (TMEM43) gene/arrhythmogenic right ventricular dysplasia. Ethical approval for this case report was obtained from the Ethical board of Public Institution Health Centre of Sarajevo Canton. In addition, the patient's parents signed consent to use information needed for this case report.

CASE REPORT

This 11-year-old female patient was admitted to Intensive care unit ventilated on Ambu ballon, Glasgow Scale Coma-6, intubated, because she experienced a cardiac arrest during training. Cardiopulmonary resuscitation (CPR) was immediately performed at the spot. VF was recorded on the ECG; she was defibrillated (100J) and returned to sinus rhythm. A magnetic resonance imaging (MRI) of the brain was done which was normal. She was on complete mechanical ventilation the first 48 h, extubated 3rd day due to favorable clinical response. Patient was then admitted to the post-intensive cardiology department with stable vital parameters. According to parents prior the accident, she was healthy, with good exercise tolerance, actively training, sometimes complained of headache and spleens. Family history: father had atrial fibrillation (AF) few years ago, so genetic testing was performed, where a mutation of the TMEM43 gene was found which is associated with arrhythmogenic right ventricular cardiomyopathy (ARVC) Type 5, as well as a variant of the calcium channel, voltage-dependent, T type, alpha 1H subunit (CACNA1H gene) which leads to familiar hyperaldosteronism Type 4. This speaks in favor of an increased genetic burden that can be associated with a disorder of cardiovascular function and a possible cumulative effect in this patient. TTE revealed a slight predominance of the left heart chambers; the right coronary artery was dilated measuring in diameter about 6.7 mm, while the origin of the left coronary artery was not visualized. Computed tomography (CT) coronary angiography with contrast confirms an ALCAPA. It was recommended that she undergo surgical correction of the congenital heart anomaly. She was also on peroral therapy: diuretics, beta blockers/propranolol, and phenobarbitone. Investigations prior arrest as a part of regular sports check-up: Cor: I_{st} heart sound normal, II_{nd} split with normal P2, systolic heart murmur I-II/VI at the 3rd and 2nd intercostal spaces. Pulse on femoral and radial arteries palpable with normal volume. 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 9 mm, IRBBB in V1 and V2, R wave in V6, no q waves, normal ST segment (Figure 1). An 24 h ECG Holter was performed before the incident, as a part of regular check-up: 99.02% QRS regular contractions were recorded, a dominant sinus rhythm, an average frequency of 91 bpm, without sinus pauses, no significant ST changes, occasional respiratory sinus arrhythmia, with rare monofocal VES (0.03%). TTE was at that time also performed: A graceful aorta with adequate flow in the ascending and descending regions of the aorta in systole as well as in the aorta abdominalis intermittent with V 98.5 cm/sec, left ventricular ejection fraction 37% (normal cardiac output).

Two months after the incident, the patient was successfully operated for ALCAPA: coronary artery transfer was performed: implantation of LCA to aorta. The patient had a good recovery, good general condition, denies any complaints since surgery, weight gain 9 kg, but further assessment of the myocardial condition is required. The possibility of ICD implantation was considered, and she was still on beta blocker (Bisoprolol).

Four months post-surgery: Sat.O_2 96%, heart rate 71 bpm, skin rosy, no rush, capillary filling time less 2 s, cor: rhythmic, pulmo eupnoeic, no restrictions, abdomen soft, no resistance, no hepatosplenomegaly, pulses palpable on all sides. ECG: SR, LAD, rate 65 bpm PQ140ms, QRS 80ms, ST isoelectric, S wave presence in V6, T neg to V3, QT 380mm, QTc 386ms; Echocardiography: normal sizes atria and ventricles, borderline normal contractility of right ventricule (RV) in speckle tracking/strain free wall-17%, normal in 3D volumetry with an EF 51% Normal systolic function of LV in 3D volumetry 64%; Lab test normal. An MRI perfusion scan of the heart was performed with question/patient: Post-CPR for VF, mutation of ARVC, what about coronary perfusion – LCA open, myocardial scars? No fibrosis and scars tissues, normal LV and RV function and size, preserved LV vitality with no arrhythmogenic cardiomyopathy criteria given (9). Computed tomography (CT) was performed, and reconstructed images revealed a giant right coronary artery originating from the right coronary sinus. The artery is dilated, has a tortuous course, is dominant, and gives numerous collaterals to the left axis deviation and the left circumflex artery (Figure 2). The left main artery (LMA) originates from the cranial contour of the truncus pulmonalis, indicating an anomalous origin of the left coronary artery from the pulmonary artery (Figure 3). Seven months after the incident an 72 h ECG Holter: Normal, sinus rhythm with an average frequency of 82 bpm; only 2 VES and one SVES were recorded - at rest. During rest and sleep are recorded, a shorter (physiological) changes in the atrial conductor without significant bradycardia (min fr. 52 bpm) and pauses. Physiological sinus tachycardia is noted during exertion, no significant

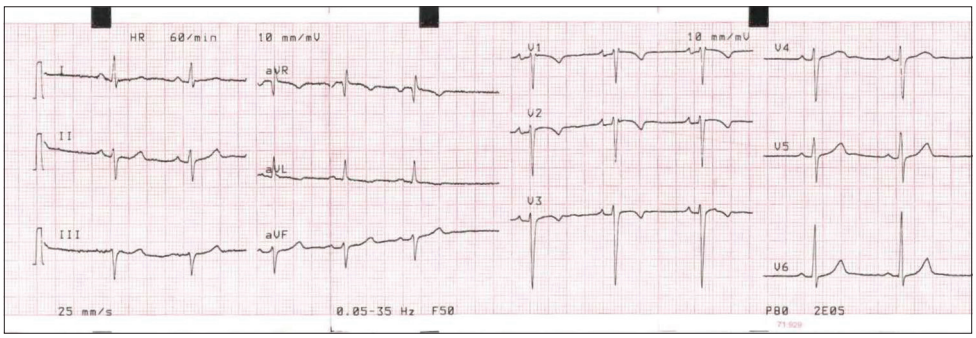

FIGURE 1. 12 leads electrocardiogram see earlier description in the text.

FIGURE 2. Computed tomography scan of the heart: Giant right coronary artery starts from the right coronary sinus, is dilated with tortuous course, dominant, gives numerous collaterals to the left axis deviation and left circumflex artery.

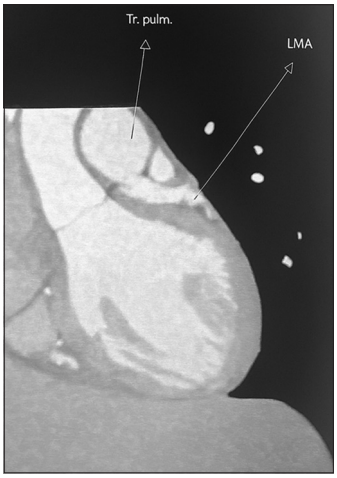

FIGURE 3. Computed tomography of the heart: Truncus pulmonalis with left main coronary artery (LMA): The LMA starts from the cranial contour of the truncus pulmonalis, that is, it is anomalous origin of the left coronary artery from the pulmonary artery.

pathological arrhythmias were found. She continues with beta blocker with the exception of heavy efforts until the stress test planned in 3 months. Further monitoring of the myocardial condition and potential fibrosis is essential, as myocardial scarring indicates an irreversible substrate for ventricular arrhythmias and recurrent malignant arrhythmias.

DISCUSSION

Individuals with ALCAPA typically present in infancy (85%) about 2-3 months after birth, as pulmonary vascular resistance drops, blood flows backward from the left coronary artery into the pulmonary artery, creating a left-to-right shunt. This reduces blood flow to the heart muscle, causing myocardial ischemia along with complications such as dysrhythmias, mitral regurgitation, and SCD. However, some individuals may have adequate collateral circulatory development, ensuring sufficient coronary perfusion like in our case report. In certain cases, the condition can progress with few or no symptoms, leading to a diagnosis that may not happen until adulthood (15%) (10). Contributing factors include right coronary artery dominance, reduced coronary artery blood flow diversion due to narrowing at the left main artery opening or a restricted pulmonary artery, and the growth of collateral vessels from the bronchial arteries supplying blood to the left coronary artery assist in extending the lifespan of patients with ALCAPA into adulthood (10). The study conducted by El-Louali et al. in 2021 shows that the median age of presentation and diagnosis of ALCAPA was 4.7 months (extremes: 7 days-10 years) (11). A study conducted by Talkhatova et al. in 2023 presents a case report on ALCAPA in an asymptomatic adult 52-year-old female patient who was incidentally diagnosed with ALCAPA during a routine medical evaluation (12). This supports the fact that the manifestation of symptoms largely depends on the development of collateral circulation, as we have already mentioned in this paragraph

In a study involving pediatric and young adolescent athletes, the majority exhibited normal ECG findings. Physiological adaptations commonly seen in athletes – attributable to the physical demands of training – were more prevalent among young adolescents and male participants. Notably, significant ECG abnormalities were infrequent, occurring in just 2.05% of cases (1.92% in children and 2.32% in young adolescents), all of which warranted further clinical assessment. Moreover, 0.27% of the cohort was diagnosed with previously unrecognized cardiac conditions solely through ECG screening. These results underscore the value of ECG as a reliable tool for identifying cardiac issues in this population (13). In the case reported by DeRose and Arcilla, a 4-year-old child – despite being asymptomatic and having well-developed collateral circulation – exhibited notable electrocardiographic findings. These included LAD, left ventricular (LV) enlargement, pathological Q waves in leads I and aVL, and ST segment depression in leads V5 and V6, indicative of anterolateral myocardial ischemia

and infarction. While pediatric ECGs may commonly display benign Q waves in inferior and lateral leads, Q waves in leads I and aVL are considered abnormal and should prompt further diagnostic evaluation (14). In our case, the only abnormality observed on the ECG was LAD with no ischemic ECG findings. While ECG screening is routinely implemented in specific populations – such as athletes – in certain countries, widespread screening for arrhythmia syndromes is generally discouraged due to concerns over false-positive results, diagnostic inaccuracies, financial burden, and limited follow-through with additional testing. Its application in pediatric and adolescent groups is especially complex, given age-related changes in cardiac depolarization and repolarization patterns (15).

Sudden cardiac arrest (SCA) in children is an uncommon event, with incidence estimates ranging from 1 to 3 cases/100,000. Most episodes occur in children who were previously asymptomatic and considered healthy. Timely recognition by bystanders, coupled with immediate CPR and access to an automated external defibrillator (AED), is often critical for survival. Accurate documentation of witnessed events plays a pivotal role in guiding both acute management and subsequent diagnostic evaluation. Following initial stabilization, ongoing care is directed by the patient's neurological and physical recovery, as well as the identification of any treatable underlying etiology. Although the potential causes of pediatric SCA are diverse, each is individually rare, necessitating coordinated care by specialized multidisciplinary teams. Preventive efforts against SCD in the young have largely centered on cardiovascular screening strategies, though SCA remains a rare occurrence in otherwise healthy individuals (15). Ventricular fibrillation (VF) leading to cardiac arrest is rare among children, accounting for roughly 10% of all pediatric out-of-hospital cardiac arrests (16). Determining the underlying cause of SCA requires a multidisciplinary approach and may remain inconclusive despite comprehensive diagnostic evaluation. Management should be individualized, incorporating family engagement and psychological support. Continued efforts are needed to improve risk stratification and to promote broader public awareness of the life-saving potential of CPR and AED training (15).

Genetic testing is gaining prominence as a cornerstone of personalized medicine, enabling the identification of pathogenic variants responsible for inherited conditions within families (15). Genetic testing is primarily indicated for individuals diagnosed with or suspected of having an inherited cardiovascular condition, or for those at elevated risk due to a known pathogenic variant within their family. In cases where a genetic etiology is suspected but no causative variant has been identified, cascade clinical screening – systematic phenotyping of family members – is generally recommended. The joint guidance from the Heart Rhythm Society and the European Heart Rhythm Association highlights the selective role of genetic testing in cardiac care. For individuals who survive out-of-hospital cardiac arrest, genetic testing is advised only when there is a clinical indication of an underlying cardiomyopathy or channelopathy; routine testing without such suspicion is discouraged. In addition, although genetic testing for AF is not currently standard practice, evolving research may justify its application in carefully chosen cases (14,17). Research conducted by Burstein et al. on children with dilated cardiomyopathy (DCM) and hypertrophic cardiomyopathy (HCM) who underwent multigene testing from 2010 to 2018 found that an increased genetic variant burden is associated with worse clinical outcomes in pediatric DCM, but this was not observed in HCM (18). In our case, genetic testing revealed mutations in the TMEM43 and CACNA1H genes, which act as genetic burdens and have a cumulative effect. These mutations can be associated with cardiovascular diseases, in our case with malignant arrhythmias-AF and ALCAPA itself.

According to the 2018 Adult CHD Guidelines by the American Heart Association and American College of Cardiology, surgery is advised for all ALCAPA patients, no matter their age or symptoms, due to the ongoing risk of ischemia, arrhythmias, and SCD. Surgical options include one-coronary or two-coronary system repair. Simple ligation, once common, is now used to delay corrective surgery in critically ill infants, allowing them time to recover before more complex procedures (10). The main aim of ALCAPA surgery is to reestablish a two-coronary artery system (19). Treatment methods involve coronary button transfer, the Takeuchi technique, connecting the subclavian artery to the left coronary artery, and coronary artery bypass graft surgery. Coronary button transfer is preferred for infants due to its excellent long-term results, while the Takeuchi procedure or bypass grafting is used for older patients when reimplantation is not feasible. Cardiac transplant is typically reserved for those experiencing significant LV failure. While surgery is the primary treatment, medications may be used alongside surgery, or in rare cases, as an alternative when surgical risks are too high (10).

A 2020 study by Muna Ismail and colleagues, spanning two decades of ALCAPA surgical cases at their institution, demonstrated encouraging outcomes. LV systolic function and chamber dimensions began to recover within 6 weeks postoperatively, achieving normalization by 1 year. Mitral valve regurgitation resolved spontaneously, paralleling improvements in LV size. In addition, somatic growth in pediatric patients showed marked improvement by 6 months following repair, and notably, all 29 patients survived. In contrast, Hu et al. reported a 1 year Kaplan–Meier survival rate of 83.4% among 80 children who underwent various surgical corrections for ALCAPA, with a median age at intervention of 7.8 months. Most received coronary artery reimplantation. The study documented 11 in-hospital deaths (13.8%) and 2 late deaths (1.3%) during the 1 year follow-up, highlighting variability in outcomes across surgical approaches and patient populations (10,20). In Mishra's study of 105 patients with anomalous coronary artery origin from the pulmonary artery – 98 of whom had ALCAPA – the median age at surgery was 5.8 months, with a median follow-up of 5.9 years. All patients underwent coronary reimplantation, resulting in an in-hospital mortality rate of 8.5%, with no deaths reported after discharge. Across the cohort, most individuals experienced improved ejection fraction, restored LV function, reduced mitral regurgitation, and relief from symptoms, regardless of the surgical technique employed. Among newborns operated on before 6 months of age, 33.6% showed marked recovery

in LV function, with mean ejection fraction increasing from 50% to 55%, and mitral regurgitation improving from moderate to mild. Conversely, 52% of those operated on later in infancy (>6 months) exhibited ongoing LV dysfunction (mean EF between 40% and 50%) and mild-to-moderate mitral regurgitation. While some of these patients improved gradually, three (3.4%) ultimately required mitral valve replacement 3 years postoperatively due to progressive regurgitation (10,21).

VF leading to cardiac arrest is rare in children, accounting for under 10% of all pediatric out-of-hospital arrests. Still, it may be related to hidden heart diseases and is often associated with improved chances of survival (16).

All adults with ALCAPA should be assessed for the risk of recurrent ventricular arrhythmias, even after full revascularization. Myocardial scars identified through late gadolinium enhancement may serve as an irreversible substrate for ventricular arrhythmias, making the consideration of an ICD for secondary prevention advisable (21). For patients who survive SCA without an identifiable reversible cause, an ICD is advised for secondary prevention (22). Long-term follow-up shows that myocardial scarring occurs in under 2% of cases, and therefore, ICD implantation is typically not advised (23). In a case report written by Jeong et al. In 2024, an 18-year-old patient who underwent surgical correction of ALCAPA in infancy experienced a ventricular tachycardia (VT) storm despite having an ICD. This was due to ischemic damage and heart muscle scars from infancy acting as the underlying cause for the VT (24,25). At present, there is no agreement on the best way to monitor VT in young patients after ALCAPA repair. This highlights the need to include specific ALCAPA follow-up recommendations in future expert guidelines (24).

CONCLUSION

Once ALCAPA is suspected due to clinical and indirect echocardiographic characteristics, direct imaging of the coronary origin should be attempted. We have to be cautious because a normal ECG does not rule out ALCAPA. This anomaly should be surgically repaired soon after the diagnosis. VF, along with other severe arrhythmias, commonly triggers cardiac arrest in individuals with ALCAPA, both before and after surgery, due to the presence of myocardial fibrosis. Children with late-onset ALCAPA syndrome, whose ECG appears normal without significant abnormalities LVH, or arrhythmias, but who show LAD and who also have a genetic predisposition to cardiovascular diseases, should maintain a lifelong follow-up care from a cardiologist with expertise in CHD. This follow-up should include surveillance for potential post-operative complications, along with regular imaging techniques to monitor their condition.

DECLARATION OF INTERESTS

Authors declare no conflict of interest.

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