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Congenital and acquired coronary artery disease in children: the challenging patients in paediatric practise

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Coronary artery disease (CAD) is a rare condition seen in the paediatric population, which makes it difficult to diagnose and manage these patients due to the broad spectrum of its clinical forms that ranges from asymptomatic course to the development of sudden cardiac death syndrome as a result of myocardial ischemia in children. CAD presents as acquired and congenital variants with varying subtypes.

Aim – to focus on aetiology, main clinical features, and early detection methods for CAD, prioritising children and adolescents to make it easier for the paediatric specialists to manage this challenging disease and prevent death.

For this review, a total of 50 articles have been analyzed from PubMed, Uptodate, Researchgate, Google Scholar, etc, which are taken from the past ten years of journals. Congenital CAD presents as defects of origin, course, or termination, and anomalous origin of the left coronary artery from the pulmonary artery, also known as ALCAPA, has the highest prevalence, and it causes 90% deaths within the first year of life. When it comes to acquired CAD, the common aetiology is found to be Kawasaki disease, which is a type of medium vessel vasculitis predominant in children between 6 months and 8 years. Another in line is familial hypercholesterolemia. It is observed that patients aged from 11–23 years, who have a history of familial hypercholesterolemia, 25% children and adolescents were found to have atherosclerotic plaques in the vessels that lead to myocardial ischemia and dysfunction. Accurate diagnosis requires specialized knowledge and skills using cardiac imaging methods. The «gold standard» method in this era is coronary computed tomography angiography, but it is not a routine test; therefore, doctors should educate themselves continuously with the latest updates in order to be able to determine the high-risk groups.

Improved knowledge about coronary artery pathologies can be of great help in diagnosing and managing children and help to prevent life-threatening complications or sudden cardiac death.

The authors declare no conflict of interest.

Keywords: coronary artery disease, congenital coronary artery pathologies, acquired coronary artery disease.

Вроджена та набута патологія коронарних артерій у дітей: складний пацієнт у педіатричній практиці

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Патологія коронарних артерій (ПКА) є рідкісним захворюванням у дитячій популяції, що ускладнює діагностику та ведення таких пацієнтів через її широкий спектр клінічних форм, який варіює від безсимптомного перебігу до розвитку синдрому раптової серцевої смерті внаслідок ішемії міокарда. Аномалія коронарних артерій може бути як набутою, так і вродженою патологією з різними підтипами.

Мета – зосередитися на етіології, основних клінічних проявах та методах раннього виявлення ПКА, надаючи пріоритет дітям та підліткам, щоб полегшити педіатрам лікування цього складного захворювання та запобігти смерті.

Проаналізовано 50 статей із реферативних баз PubMed, Uptodate, Researchgate, Google Scholar за останні десять років. Вроджені аномалії розвитку коронарних артерій проявляються у вигляді дефектів відходження, ходу або закінчення артерій, серед них аномальне відходження лівої коронарної артерії від легеневої артерії, також відоме як ALCAPA, має найбільшу поширеність і є причиною 90% смертей протягом першого року життя в таких дітей. Щодо набутої ПКА, то найпоширенішою причиною є хвороба Кавасакі, яка є різновидом васкуліту судин середнього калібру та переважає в дітей віком від 6 місяців до 8 років. Наступною в цьому переліку є сімейна гіперхолестеринемія. У пацієнтів віком від 11 до 23 років, які мають в анамнезі сімейну гіперхолестеринемію, у 25% дітей та підлітків були виявлені атеросклеротичні бляшки в судинах, що призводять до ішемії та дисфункції міокарда. Діагностика ПКА вимагає спеціальних знань і навичок із використанням сучасних методів візуалізації серця. «Золотим стандартом» нині є коронарна комп'ютерна томографія-ангіографія, але вона не є рутинним дослідженням, тому лікарі повинні постійно підвищувати свою кваліфікацію, щоб мати змогу визначати групи високого ризику.

Поглиблення знань про ПКА може стати в нагоді в діагностиці та лікуванні дітей і допоможе запобігти небезпечним для життя ускладненням або раптовій серцевій смерті.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: аномалії коронарних артерій, вроджена патологія коронарних артерій, набута патологія коронарних артерій.

Introduction

Coronary Artery Disease (CAD) in children is a condition that occurs due to anomalies seen in the normal course of the coronary arteries. CAD can be divided into two types, congenital and acquired depending upon the cause affecting the coronary arteries. It results in the development of ischemia and later myocardial infarction (MI), which can be the cause of sudden cardiac death (SCD) in both adults and children. Though MI is rare <1% among children of all age groups, it can still present dramatically in paediatric practice, predominantly in children with anomalous origin of left coronary artery from pulmonary artery, also known as ALCAPA [9]. The reason that makes this pathology challenging to diagnose is its wide clinical polymorphism because its course can range

from an asymptomatic form or an otherwise healthy appearing child to the earliest manifestation being SCD. According to a 7-year study done by the European Society of Cardiology, 1.1 children per 100000 per year died of SCD in Denmark [28]. Another study suggests that 0.2–0.5 deaths per 1000 live births occurred in infants of 2–3 months of age, and the most common cause was cardiac diseases, out of which CAD bags the second or sometimes third position [28]. As mentioned before, mostly the course of this disease can be asymptomatic, but sometimes syncope without any known reason can also occur. Statistics show that 371 paediatric patients admitted in a hospital due to syncope, 2.2% of them had congenital CAD [14]. A meta-analysis conducted among 2890 athletes and 2170 non-athletes aged ⩽35 years who died of SCD also revealed that the aetiology of death among non-athletes was CAD, which was thought to be expected from athletes [7].

According to a source from the National Library of Medicine, the most common aetiology of acquired CAD is Kawasaki Disease (KD). It is a well-known fact backed by the evidence that this condition affects children predominantly under 5 years of age; more boys than girls which is 1.5:1. The first reported case was in the 1960s by Dr. Tomisoku Kawasaki, who monitored 50 patients with KD and came to a pioneering conclusion that CAD and coronary artery aneurysms are the significant complications of KD with severe consequences in adulthood [23].

Acquired CAD is also associated with Familial Hypercholesterolemia (FH), which is a monogenic disease and is accompanied by an increase in low-density lipoprotein cholesterol levels from birth. Due to this, atherosclerosis in the coronary artery (CA) may occur, leading to ischemia of the myocardium and increased risk of cardiovascular problems, which can begin in childhood and present in adult patients. L. Shoa et. al. from Stanford University found that patients who were FH carriers had increased risk for CAD with an odds ratio of 1.53 (95% CI 1.24–1.89) [5,15].

Even though there are many studies, due to a lack of experience and clinical data, diagnosing CAD in children is a very cumbersome process. Its rare oc-

Table 1

Comparative characteristics of aetiology and clinical presentation of congenital and acquired coronary artery disease

Feature	Congenital Coronary Artery Disease	Acquired Coronary Artery Disease
Definition	Abnormalities that are present from birth due to various unknown reasons	Abnormalities that occur after birth due to previous pathologies
Aetiology	1) Anomalies of origin: – Origin of CA from PA. – Anomalous origin of CA from Aorta. – Presence of only 1 CA. –Ostium abnormality, displacement of coronary ostia. –Anomalies of course: a) malignant course Interarterial. b) benign course Intramural. Retrocardiac. Retroaortic. Prepulmonic. 2) Anomaly of the CA vessel wall. 3) Anomalies of Termination. 4) Anomalies associated with congenital heart disease (CHD)	1) Kawasaki disease (inflammation of arteries) 2) Familial hypercholesterolemia (elevated cholesterol levels) 3) Infections (e.g., bacterial or viral myocarditis, endocarditis) 4) Rheumatological conditions (e.g., systemic lupus erythematosus, Juvenile idiopathic arthritis) 5) Other factors such as Obesity, Type 2 Diabetes, Inborn errors of metabolism
Clinical manifestations	1) Often asymptomatic. 2) If symptomatic, can present with: – Syncope – Presyncope – Fatigue – Angina - Poor exercise tolerance. – severe myocardial dysfunction due to myocardial ischemia, signs of shock or murmur of mitral regurgitation	- Angina (chest pain) – Dyspnoea (shortness of breath) – Failure to thrive (in children) – Symptoms of Kawasaki disease (fever of unknown origin for more than 5 days, bilateral conjunctivitis, mucositis, lymphadenitis, oedema of hands and feet). – Symptoms of myocarditis (fatigue, flu-like symptoms) – Tachyarrhythmias (supraventricular tachycardia or ventricular tachycardia in early adolescence)

Notes: PA – Pulmonary Artery; CHD – Congenital Heart Defects.

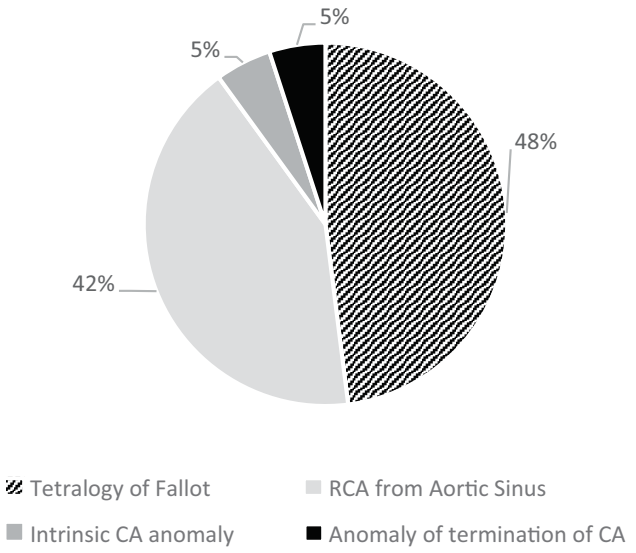

Fig. Prevalence of different types of congenital CAD

currence makes it a difficult pathology to diagnose in time. Therefore, proper diagnostics and screening should be done in order to obtain the pathologies of CA early. The ***aim*** here is to focus on aetiology, main clinical features, and early detection methods for CAD, prioritising children and adolescents to make it easier for the paediatric specialists to manage this challenging disease and prevent death.

The study for this review is sourced from PubMed, UpToDate, Researchgate, Google Scholar, and many other trusted organisation's websites. It focuses on different guidelines, meta-analyses, and cohort studies done on CAD, MI in children, and the cause of SCD. The total of 50 articles of the past 10 years of publishing have been analyzed for this purpose, out of which 31 of utmost importance have been listed here. The sources are in English, Ukrainian, and Chinese. Keywords used were «coronary artery disease», «congenital coronary artery pathologies», acquired coronary artery disease», «ALCAPA», «Sudden Cardiac Death in children», «Kawasaki Disease», «Myocardial Dysfunction», «Myocardial Infarction», «Familial Hypercholesterolemia», «Children».

CAD is the pathology of blood vessels that supply blood to the heart, which results in decreased blood circulation in the heart muscle, causing ischemia of the myocardium. It is of two types, namely congenital CAD and acquired CAD, which are listed in Table 1 [7,16,20,24].

Congenital CAD, precisely abnormal aortic origin of CA, is found to be present among 0.64% of total births. Out of those 0.5–3% children have an anomaly of CA arising from the opposite sinus of Valsalva, and among them, 20% lead to complications such as arrhythmias or MI [6]. Anomalies such as congenital CA aneurysm, myocardial bridge formation, congenital CA stenosis, abnormal number of ostia or left CA (LCA) or right CA (RCA) or both CA arising from right aortic sinus are a part of it. All of these create a hindrance in the normal path of the blood to the myocardium [29]. Among the most prevailing congenital CADs is ALCAPA, also referred to as Bland-White-Garland syndrome. ALCAPA occurs in every 1 in 300,000 children, accounting for 0.5% of CAD, and it causes 90% deaths within the first year of life, if surgery is not done on time [6,11]. It decreases the flow in the left myocardium, and usually within 4–6 weeks of life, the child presents with diaphoresis, poor feeding, and failure to thrive, which may be indicative of poor perfusion due to less oxygenated blood supply, indicating pulmonary origin. However, not all patients die within a year of life; some cases have also been reported in adolescents and young adults. This is due to collateral formation and development of «steal phenomenon» i.e., more blood flow through the PA robbing the left myocardium of blood, causing ischemia. This causes angina, arrhythmias, and insufficiency of the myocardium, leading to SCD [6].

CAD can appear as a single major defect in children or can also be accompanied by other cardiac anomalies. Munevver Tugba Temel et al. [25] from Gaziantep University, Turkey did a study on 515 patients with CHD undergoing angiogram, which concluded that 42 patients between the age of 5.3±2 years confirmed for CAD; 90% of those patients had anomalies of origin; 4.8% had anomalies of termination, and 4.8% had anomaly of intrinsic CA. Among two-thirds of the patients, 42% were found with anomalous origin of RCA and 48% had Tetralogy of Fallot (TOF). A diagrammatic representation of the same is shown in (Figure) [11].

As described before, CA pathologies have also been associated with Tetralogy of Fallot (TOF), one of the most common congenital heart diseases, which manifests with cyanosis and «Tet spells» almost after a month a child is born. Anyhow, the patients with TOF have to undergo surgery within a year for better survival, but the presence of CAD along with it can make the management strategy more tedious for doctors. This fact was proven by the team of cardiologists from Leiden University Medical Centre in the Netherlands that came to the conclusion that CA

discrepancies were observed in 6% TOF patients, within which abnormal course of CA is more prevalent, with the estimate of 72%, which crosses the right ventricle outflow tract, and 28%, which have retroaortic course [20]. Coronary arteries that have an interarterial course due to TOF are potentially more life-threatening, as there is a huge chance that during exercise, the blood flow in them can get obstructed due to increased pressure in the aorta and PA from both sides, posing a risk of SCD [20]. Treatment of these pathologies requires reimplantation of CA, a baffle reconstruction, but even that does not guarantee survival. It has also been reported by the European Society of Cardiology that at any age, the cases of SCD also tend to be more in males than in females, 1.7:1, who have had surgical correction for CAD in childhood [30].

When it comes to acquired CAD, the common aetiology is found to be Kawasaki disease, which is a type of medium vessel vasculitis predominant in children between 6 months and 8 years. Different studies show that the prevalence of KD is 5-fold higher in Japanese children than in Caucasians, Hawaiians, and Filipinos and patients from Western countries [20]. The forementioned pathology is responsible for myocarditis, acquired valve disorder, dilation of the aortic root, and the most important for prognosis in this category is CA involvement due to CA aneurysms in children [29]. If the diagnosis is delayed and treatment is suspended for more than 10 days, KD can significantly increase the risk of CAD. This was proven by the group of Chinese paediatric cardiologists from Beijing, they studied 1052 patients of KD during a period of 5 years between the age group of 2 months to 13.8 years and found that the children who got intravenous immunoglobulin treatment early i.e. within 1–4 days showed lesser risk (17.6%) of CA complications than those who were treated late i.e. after 10 days of fever with higher risk (33.7%) [29]. Children who were treated for KD predominantly of age <6 months were found to be more prone to coronary artery aneurysms [22].

Another cause of progressive CA damage is FH, which is due to abnormal cholesterol metabolism, causing atherosclerosis in children and often premature death. FH is an autosomal dominant condition that presents as an increase in low-density lipoprotein cholesterol level in children. In a study done by doctors of the Children's Health Hospital in Ireland, it was observed that between 11 and 23 years of age, 25% children and adolescents with FH were found to have atherosclerotic plaques in CA. This condition was found to be more common in homozygous FH allele-containing individuals than heterozygous [13]. The homozygous individuals have a poor prognosis and are more likely to pass away from CAD. Usually, there are no significant symptoms to accommodate in the specific bracket for discussion on FH, but at times, xanthomas, xanthelasmas, which are cholesterol deposits around the eyelids, xanthomata commonly in the Achilles tendon or dorsum of the hands, and chest pain during activity can be observed. If these symptoms lead to acute coronary syndrome within a year, then there is 2 times increased risk of SCD even after the treatment from statin therapy is done [26].

Due to such dire consequences, it has become the need of the hour to formulate the diagnosis on time. Objective findings, general symptoms and taking a thorough history of the patient is of paramount importance as possible clinical presentation of CAD varies in different age groups; among infants it can be poor feeding, heart failure; among older children it can be pain in the chest, dyspnoea or syncope; among teenagers SCD after a strenuous exercise can be the earliest symptom [12]. However, these findings are not very discrete, and it is often too late until they present in a patient, so in order to diagnose the child with a possible risk of CAD before he/she is symptomatic, screening should be done for the risk groups. They can be classified as high-risk groups such as children with family history of hypercholesterolemia, CHD, KD, Diabetes Mellitus, recreational athletes, competitive athletes; moderate-risk groups for instance, heterozygous FH, history of episodes of syncope and angina without specific findings even after ECG, severe obesity and low-risk groups being cancer patients, other inflammatory diseases (Systemic Lupus Erythematosus) etc. to name a few [3,9].

In general, work-up for FH Universal screening is done twice, once between 9–11 years and then between 17–21 years, and Cascade screening is done for children with premature atherosclerotic cardiovascular disease or very high cholesterol at 2 years of age [8]. The screening for KD can also be done for inositol 1,4,5-triphosphate 3-kinase gene or miRNA (miR-223), which is responsible for the development of CA lesions [17]. Next is exercise stress electrocardiogram for both symptomatic and asymptomatic patients, a positive stress test at low workload sug-

Table 2

Summary of important paediatric study with CAD as the cause of life-threatening conditions in children

Year	Source	Key findings
2017	AHA report on Kawasaki disease	25% of children diagnosed with Kawasaki disease at age 5 or younger, if untreated, develop CA aneurysm, a leading cause of acquired CAD [21]
2019	Radiological Society of North America report	10% of autopsy reports of patients who died from MI had a history of familial hypercholesterolemia, which caused CA Insufficiency leading to MI [19]
2019	USCDC report on sudden cardiac arrest in children	392 children died from cardiac arrest without any prior diagnosis of a health condition. The autopsy revealed CAD [27]
2022	Report on congenital anomalous origin of CAD in children with syncope	2.2% of children with syncope were found to have CA anomalies [14]
2023	World Journal of Cardiovascular Disease report	1 in 300,000 patients has ALCAPA, which is a form of CAD [4]

gests ischemia and is indicative of invasive CA angiography, and at high workload allows the doctor to perform additional tests, such as coronary computed tomography to be sure of the state of CA [1]. Another screening method is the coronary artery calcification score using non-invasive coronary computed tomography angiography to check for narrowing of coronary arteries [2,3]. If the patient is symptomatic or screening tests are positive, other instrumental or invasive methods are used to make the diagnosis unequivocal. First being Electrocardiogram**,** in symptomatic patients, signs of ischemia, abnormal Q-wave in leads aVL, V4-V6, and ST-T abnormality are more common; this test should be done in athletes as a regular follow-up, in children suspected of FH and KD. But due to its low sensitivity up to 60% and specificity up to 90% its accuracy is debatable [25]. Transthoracic Echocardiogram (TTE) gives information about the position of CA, dilation of CAs, extensive collateral formation between RCA and LCA, aneurysm formation, etc. TTE is also the investigation of choice in CAD detection due to its no radiation exposure, non-invasive, cost-efficient, and easy implementation methods, and it should be made mandatory in obese people, infants with TOF, diabetic population, or in whom ECG was inconclusive. It has a sensitivity of 89%, specificity of 96% [12]. Coronary Magnetic Resonance Angiography is used for probable diagnosis of anomaly of CA from the opposite sinus, which is also an early factor for SCD, and can be an investigation for patients with CHD. It also identifies CAD in children with KD. It also has 76% more accuracy than an ultrasound. It gives a sensitivity of 97.5% and specificity of 83.5% [10]. Next is Coronary Computed Tomography Angiography, this high imaging modality is the «gold standard» investigation due to its non-invasive nature, high speed accuracy, and excellent sensitivity up to 99% and specificity up to 92%. Children presenting with non-traumatic chest pain, history of KD or ALCAPA should undergo this test [12]. A few modern studies formulated the common hypothesis that the number of CA anomalies was better detected through coronary computed tomography angiography. This theory was proven by the largest clinical data, citing that the overall prevalence, which was confirmed from this technique, was more than three times higher (7.85% versus 2.02%; $p<0.01$) as compared to invasive coronary angiography [18].

CAD poses a great burden not only on family members of the patients but also on the doctors involved in treating the child because of the broad horizon of its clinical manifestations. These life-threatening conditions almost always develop suddenly in previously healthy children; that's why it's important to pay significant attention to medical history (FH, DM type II, KD), history of syncope, chest pain, ST-T changes in ECG, specific OR non-specific.

One of the examples of how CAD can occur due to diverse reasons is shown in Table 2.

Taking into account all the problems about the management of CAD, the solution is to find screening tests that are cheap, widely available, and backed with high sensitivity and specificity. So, TTE being cost-effective than others is widely used, but due to the imaging quality of an ultrasound, it sometimes makes the results deceitful in cases like ALCAPA. Coronary computed tomography angiography also has one drawback, which is high radiation exposure in this technique, which makes it quite dangerous for children, especially neonates and infants. So, the use of these techniques should be limited and done only if and when necessary, depending on the judgement of the doctors. There is no use for ambulatory ECG and specific blood test for biomarkers of CAD as they cannot give conclusive data, so they can only be used

to rule out other pathologies and make differential diagnosis [3]. Therefore, if the doctor suspects CAD, it is necessary to do further tests with high-resolution imaging that has a high accuracy rate.

Conclusion and prospects for further developments

CAD in paediatric practice still remains a challenging problem to tackle for doctors due to its less frequent occurrence in children than in adults, often asymptomatic course of presentation, and less awareness among paediatricians, resulting in a high probability of misdiagnosis. On the other hand, it stands on the second or third position among all causes of SCD in athletes and non-athletes. Therefore, this makes it necessary to disseminate information among the doctors of different paediatric specialities so that they are able to detect CA anomalies early and prevent life-threatening complications. This can be achieved by doing screening tests for the risk groups and using the «gold standard» modalities on patients with high suspicion to make them less prone to complications and provide timely care. Once CAD is suspected, a full workup should be done by the doctors. Regular follow-ups, timely screening, and early identification of the risk factors are the key to controlling CAD, especially in cases of KD, FH, and CHD.

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