Review Article

Myocardial Infarction in Adolescents: A Growing Concern – A Narrative Review

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ABSTRACT

Background: Myocardial infarction is the leading cause of mortality worldwide. Although it is believed to be uncommon in teenagers, current research indicates that the prevalence of myocardial infarction and its associated consequences is rising in this age group, potentially linked to increasingly hazardous eating and lifestyle choices. Objective: To evaluate and include several recent studies that identify the key differences in risk factors, causes, and clinical manifestations of myocardial infarction in older adults and adolescents, highlighting the growing prevalence of myocardial infarction in adolescents and its unique management strategies. Methodology: Data was collected and analysed following a review of relevant studies published in journals indexed in PubMed, Google Scholar, and Science Direct. Results: Clinical presentation of myocardial infarction is similar in adolescents and older adults. While a positive history of angina, diabetes, and hypertension have a greater impact on adults, a positive smoking history and family history of myocardial infarction are more directly linked to myocardial infarction in younger individuals. Most adolescents present with ischemic discomfort related to congenital abnormalities or acquired changes due to Kawasaki disease, improper medication, and substance use. Primarily, many may have a family history of early coronary heart disease or a history of arterial or venous thrombotic events, which may contribute to their presenting condition. Adolescents and young adults are more likely than middle-aged and older individuals to experience ventricular tachycardias and peak creatine kinase levels. Additionally, adolescents tend to have a better prognosis. Conclusion: There is a higher prevalence of myocardial infarction in adolescents, and notable distinctions exist between adolescents and older individuals regarding their presentation, investigation results, and risk factors for myocardial infarction.

Key words: Adolescents, myocardial infarction, clinical manifestation, and outcome/prognosis

yocardial infarction (MI), commonly known as a "heart attack," is caused by a partial or complete blockage of the myocardium's blood supply [1]. Myocardial infarction is the world's leading cause of death. However, acute MI is becoming more common in young people. Contrary to popular belief, acute myocardial infarction (AMI) is not likely to be the cause of chest pain in adolescents. Hence, this specific patient group are predisposed to a significant risk of experiencing a severe result. There are two possible causes of acute MI in adolescents: early atherosclerosis or non-atherosclerotic causes, including cocaine-induced vascular spasms [2].

Acute myocardial infarction (AMI) is less common in young adults and adolescents than in older people. It is a serious

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problem for the patient and the treating physician since these younger people have distinct risk factors, clinical presentations, and prognoses than older persons [3]. While adults develop coronary artery disease (CAD) as a result of lifetime deposition of atheroma and plaque, which causes coronary artery spasm and thrombosis, adolescents frequently experience either an acute inflammatory condition of the coronary arteries or a malformed origin of the left coronary artery (LCA). Myocardial infarction (MI) can occur during pregnancy, often in combination with coronary artery stenosis [4]. Kawasaki disease and aberrant left coronary artery originating from the pulmonary artery (ALCAPA) are the two most frequent causes of paediatric myocardial infarction (PMI) [5, 6].

The majority of young individuals who have acute myocar

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-dial infarction (AMI) present with chest pain, and their clinical presentation is comparable to that of older patients. In young people, the diagnosis of acute MI is more likely to be delayed because of a reduced suspicion of the condition and atypical presentation in certain patients [7, 8]. Despite earlier diagnosis of MI in older patients, it was found that they are more likely to be hospitalised. Further, it is seen that the hospital mortality in older individuals with MI is higher as compared to young adolescents [9].

While several conventional risk factors, including hypertension, diabetes mellitus, dyslipidemia, or atrial fibrillation, are frequently present in older patients with MI, a significant difference is seen in younger MI patients. People in this group who had smoked in the past and had their first MI earlier in life are more likely to present with MI [10]. It is seen that young adolescents with MI are correlated with more modifiable risk factors like substance use, obesity, etc. [11]. According to one study, younger AMI patients are more likely than older persons to have a substantial family history of early CAD [12]. Furthermore, the standards for electrocardiograms, echocardiograms, and enzymatic tests have been wellestablished in adults, but there are still some difficulties in detecting MI in children.

Age-related changes in heart structure may sometimes cause problems with the electrocardiographic diagnostic criteria for ischaemia [13]. Adolescent myocardial infarction can be prevented and detected early with the aid of a better knowledge of the prevalence and progression of AMI in this age range. This review's goal is to examine and incorporate a variety of current studies that are pertinent to determining the main distinctions between the clinical manifestations, aetiology, and risk factors of myocardial infarction in older people and adolescents.

METHODOLOGY

Data from papers published over the last 50 years was compiled in this review. Relevant publications from 1969 to 2023 were found from database searches such as PubMed, MEDLINE, Embase, and other pertinent sources, including Google search engines and World Health Organisation websites. Table 1 lists the search terms that were utilised and provides an example of a database's search technique.

ITEMS	SPECIFICATIONS
The date of the search (along with the month and year)	21 October 2023
Other sources and databases were searched.	A systematic search of PubMed, Embase, MedRxiv, EBSCO MEDLINE, and Scopus electronic databases is done.
MeSH and free text search	For published publications,

ITEMS	SPECIFICATIONS
phrases and filters were among the search terms utilised. Note: Please provide a complete search strategy for one database as an example using an independent supplement table.	medical topic headings (MeSH) and free-text phrase keywords such as "myocardial infarction," "adolescents," "heart attack," and "atherosclerosis" will be utilised.
Timeframe	6 months
Criteria for inclusion and exclusion (kind of study, language limitations, etc.)	Includes free review articles narrowed down after selection process based on relevance to title and aim of review and also includes free full text published researches written in the English language Excludes book chapters, paid articles, meta-analysis and articles published in languages other than English
Procedure for selection (who made the choice, if it was done separately, how consensus was reached, etc.)	All authors conducted the selection process was carried out by all writers after debate. the selection following discussion
Additional factors to take taken into consideration, if applicable	Not applicable

RESULT AND DISCUSSION

In adults, coronary artery disease (CAD) is the most prevalent cause of myocardial infarction (MI). [14] Younger individuals with MI can be divided into two groups: those with coronary artery disease from different sources and those with angiographically normal coronary arteries. pathophysiology that underlies both groups is quite similar, notwithstanding this difference. The etiology of MI varies when coronary arteries show up as normal on angiography. This condition may be caused by coronary artery thrombosis, embolization, spasms, or a mix of these. [15].

Although pediatric antiphospholipid syndrome (APS) has been studied little, it has the potential to cause serious morbidity in children. Although the exact mechanism of APS pathophysiology is yet unknown, anomalies have been observed in the complement cascade, platelets, neutrophils, endothelial cells, and monocytes [16]. This disorder is linked to recurrent arterial and venous thrombosis and usually strikes people at a younger age. Persistent pathogenic antiphospholipid antibodies (aPL) are present when it happens [17]. By obstructing the coronary arteries, these thrombotic episodes may result in a myocardial infarction [18]. Furthermore, as anticoagulant therapy decisions can significantly affect the child's and their family's way of life, they must be carefully

considered for young children with the condition. In the context of pediatric-onset APS, there is a dearth of knowledge about therapy and long-term outcomes, with the consensus being based on a small number of studies and case reports. [19].

One extremely uncommon cause of myocardial infarction is nephrotic syndrome. However, some conditions, such as dyslipidemia, abnormalities in the fibrinolytic system, and low anticoagulant levels, can cause hypercoagulability. A lack of antithrombin III is one of the primary causes of hypercoagulation in nephrotic syndrome [20]. Due to congenital defects, some people may have their first myocardial infarction in their early adult years. One such aberration is myocardial bridging, which can result in MI during systolic contraction and occurs when coronary arteries are lodged in the myocardium underneath muscle layers. [21]. The left circumflex (LCX), which originates from the right sinus of Valsalva (RSV), is the most common anomaly in congenital aberrant origin of coronary arteries. Other variations include both coronary arteries from RSV, the left anterior descending coronary artery from RSV, and one coronary artery from the left sinus of Valsalva. Although abnormal left main coronary artery (LMCA) starting with RSV is rare, it carries a high risk of sudden cardiac death. Myocardial ischemia in young individuals is recognized to be caused by the uncommon congenital defect known as ALCAPA syndrome [22].

The majority of misdiagnosed patients die during the first year of life, although the degree of collateral circulation that forms between various coronary artery areas is linked to survival after infancy [22]. Although ALCAPA syndrome seldom manifests in late childhood, adolescence, or adulthood, it may play a substantial role in sudden cardiac arrest [23]. Because pediatric patients frequently have rapid heart rates, doing a CT scan might provide technical difficulties, requiring the addition of coronary angiography to the examination. Achieving two coronary system perfusions should be the main goal of surgical intervention [24]. In one instance, a 10-yearold girl was admitted to the hospital right after suffering a myocardial infarction. The echocardiogram revealed that the left coronary artery was not coming from the pulmonary artery, which raised suspicions of an abnormal origin of the left coronary artery from the pulmonary artery (ALCAPA). A CT scan and cardiac catheterization were used to confirm the diagnosis later on [25].

Myocardial ischemia is known to be caused by the anomalous origin of the right coronary artery (ARCA). This is another congenital condition that can cause myocardial infarction and angina pectoris in young people. It is also a significant contributing factor to young athletes' unexpected cardiac deaths. This condition is typified by an abnormal relationship between the left coronary sinus and the right coronary artery, which may lead to myocardial ischemia and, in some cases, sudden death [26]. During coronary angiography, CT coronary angiogram (CCTA), or autopsy, the

patient's presenting symptoms are frequently used to make the diagnosis of CA [27]. As a result, CCTA is currently the gold standard imaging method to identify the etiology and development of CA [28]. In cases of ARCA with evidence of ischemia, surgical revascularization is a class I recommendation, according to the joint recommendations of the American Heart Association and the American College of Cardiology [29].

SCAD seldom causes acute myocardial infarction in young to middle-aged individuals, but it is becoming more wellacknowledged. It happens when an intramural hematoma-filled false lumen forms as a result of a rip in the inner layer of the artery. The real coronary lumen may be compressed by the hematoma's rising pressure, limiting blood flow and leading to coronary insufficiency [30]. Elevated cholesterol levels are a hallmark of the autosomal dominant disorder known as familial hypercholesterolemia. In young people, it may result in myocardial infarction (MI) and early atherosclerosis. Two percent of boys aged 15 to 19 had advanced coronary atheroma, according to a research of 760 deaths from accidents, suicide, or homicide that involved people aged 15 to 34. [31]. An increased risk of coronary heart disease and atherosclerosis has been linked to elevated total plasma homocysteine levels. Young people can develop atherosclerosis due to elevated homocysteine levels, which can be caused by genetic defects, vitamin B deficiency, certain medicines, and renal impairment. Increased oxidative stress, decreased endothelial function, and a risk of thrombosis are the causes of this [32].

Homocysteine can influence preclinical morphology and function and contribute to the development of cardiovascular disease through a variety of unique mechanisms, such as its harmful effects on smooth muscle cells and vascular endothelium [33]. According to research, over half of kids between the ages of 10 and 14 showed some signs of atherosclerosis. Additionally, in American youngsters killed in auto accidents, lipid-filled macrophages were found in the coronary arteries and aortic inner layer [34]. Atherosclerosis begins to develop in the womb, progresses through puberty, and finally shows clinical symptoms in adulthood. In addition to avoiding the formation of new atherosclerotic plaques, adolescents who get appropriate pediatric care may be able to stabilize existing ones [35]. According to histological research, plaque formation and maturation gradually increase from infancy through adolescence and beyond into maturity [34].

A physical examination, non-invasive diagnostics, and pertinent family history can all be used to determine which youngster is at risk. ASCVD risk factors must be addressed, and Low-density lipoprotein-cholesterol (LDLc) levels must be below 110 mg/dl. A goal LDLc of fewer than 100 mg/dl is advised in the case of diabetes. By screening and acting during the early phases of adolescence, pediatricians may address this public health risk in a rational and optimistic manner, as atherosclerosis is typically a quiet illness in children [36].

Myocardial infarction might occur as a result of the numerous cardiovascular and hematological consequences of cocaine use, such as coronary vasospasm, hypercoagulability, and elevated sympathetic activity [32].

Additionally, it has been demonstrated that cocaine dosage reduces left ventricular performance and increases end-systolic wall stress [37]. Cocaine increases myocardial demand by increasing heart rate, blood pressure, and contractility. According to research, 48% of young patients with non-traumatic chest pain who were brought to the emergency room had cocaine usage. [38]. Cocaine induces myocardial ischemia or myocardial infarction (MI) through a multifaceted array of mechanisms, including increasing myocardial oxygen demand via elevated heart rate, blood pressure, and contractility; diminishing oxygen supply through vasoconstriction; activating platelets and perturbing the balance of procoagulant and anticoagulant factors; and accelerating the progression of atherosclerosis

In affluent countries, Kawasaki disease is one of the leading causes of acquired heart disease in children [39]. In children under five, it presents as a feverish sickness accompanied by mucocutaneous abnormalities [40]. Thrombosis or stenotic lesions associated with the aneurysms may occur in those who have coronary artery damage due to the condition, increasing their risk of myocardial infarction, sudden death, and congestive heart failure. [41, 42]. Approximately 25% of individuals with known instances of Kawasaki disease developed coronary aneurysms, with 55% of these cases showing regression over time, according to a 1973–1983 cohort study carried out in Japan that followed up on these patients over 10–21 years. During the follow-up, they observed that 4.7% of patients had ischemic heart disease, 1.9% experienced myocardial infarction, and 0.8% experienced death [43].

Adolescents may get acute MI due to early atherosclerosis or for other causes, such as cocaine-induced vascular spasms [44]. The early onset of coronary atherosclerosis is linked to the existence of conventional cardiovascular risk factors. For instance, smoking, hypertension, dyslipidemia, obesity, inactivity, and stress [45]. One of the most significant risk factors is smoking since it is linked to low-level cholesterol deposition, platelet aggregation, increased vascular endothelial cell damage, and vascular occlusion. People with a genetic predisposition, such as those with hereditary hypercholesterolemia or mutations in the factor V Leiden gene, are also at a higher risk of MI brought on by early atherosclerosis [46].

Unconventional risk factors, including pregnancy and direct contact sports, are linked to several underlying diseases, including MI from nonatherosclerotic causes [47]. Cocaine use and excessive drinking are also connected to myocardial infarction, tachyarrhythmias, and bradyarrhythmias. These conditions are linked to increased systemic blood pressure, heart rate, oxygen demand, and vasoconstriction [20, 48].

Among the risk factors for myocardial infarction in young individuals are autoimmune conditions such as rheumatoid arthritis and Systemic Lupus Erythematosus (SLE) [20]. Men are more likely to have MI in younger age groups, but as people age, fewer male patients with AMI are admitted to hospitals, and as the number of female patients rises, the sex ratio decreases [49].

The decrease in oestrogen and its cardio-protective effects in elderly women may be one of the possible explanations of this [50]. Smoking, obesity, and a family history of coronary heart disease were more common among young patients who were suffering from acute myocardial infarction (AMI). Nonetheless, the prevalence of dyslipidemia, diabetes mellitus, and hypertension was the same for both young and elderly AMI patients. The low frequency of smoking among the elderly can be explained by the fact that postmenopausal women, who are generally non-smokers, become more prevalent in the old population with AMI and that the majority of elderly persons quit smoking as they age. Age itself is a risk factor, according to similar research [51]. Since young patients with MI are rare, a thorough diagnosis is necessary to rule out other possible causes.

Adolescents with MI are prone to experience ischemic chest discomfort, which may be attributable to congenital diseases such as coronary artery abnormalities, aortic valvular and supravalvular stenosis, or acquired conditions, including Kawasaki illness, substance addiction, and medication [52]. It's important to record recreational drug use, ask about family history of early coronary heart disease (CHD), and evaluate risk factors, including smoking, obesity, diabetes, and dyslipidemia, while evaluating a young patient's medical history.

It's also important to record any prior episodes of recurrent arterial and venous thrombosis that could be pertinent in this situation [45]. Even the clinical characteristics of MI can occasionally be vague, leading to confusion with myocarditis. However, research has shown that the majority of patients with myocarditis are older than those with MI, and an endomyocardial biopsy can be performed to confirm this [53]. There are now research being undertaken to better understand the absence of distinctive traits for this age group, which can be primarily ascribed to the lack of prevalence of MI cases that have been analysed.

Younger people's initial therapy for MI is a little different from adults. The history of MI in adolescents includes spasms caused by cocaine or drug use; in this case, thrombolysis should be administered following the administration of vasodilators if symptoms do not resolve right away and if there are no changes in the ST segment. These patients should continue taking nitrates to prevent coronary spasms [20]. Primary angioplasty is superior to thrombolysis for patients with myocardial infarction (MI) brought on by accelerated premature atherosclerosis [54]. Percutaneous transluminal coronary angioplasty is taken into consideration in situations with

myocardial bridging and spontaneous coronary artery dissection [21, 55]. In most situations, antiplatelet medications like aspirin are advised.

Anticoagulation should be taken into consideration in nephrotic syndrome instances where blood albumin levels are less than 20 g/l [56]. Beta blockers should be avoided in situations of cocaine or amphetamine-induced coronary spasms because they may exacerbate the spasm [20]. Evaluating subgroups of patients with acute myocardial infarction is a popular method for determining prognosis and identifying high-risk groups who might profit from intense diagnostic and therapeutic measures [57]. Young patients had extremely low one-year mortality and inpatient mortality rates. Despite significant myocardial infarctions, this remarkable prognosis was noted, and the cardiothoracic ratios and ejection fractions were comparable to those of elderly individuals. On the other hand, older patients exhibited greater multisystem illness symptoms, a history of myocardial infarctions, congestive heart failure, angina pectoris, worsening heart failure, and atrioventricular block [58].

Patients who were monitored for a year also had a reduced post-discharge death rate than middle-aged and older patients. Nonetheless, among patients released alive and monitored for a year, the non-fatal reinfarction rates in the three groups were nearly identical. When all coded problems were included, complications were less common in younger patients. Shock, chest pain, and Congestive Heart Failure (CHF) are serious adverse effects. Younger patients had a considerably lower likelihood of being prescribed digitalis, diuretics, and antiarrhythmic medications upon discharge compared to middle-aged and older patients. On the other hand, compared to their older counterparts, young and middle-aged people were more likely to be released with 3-blockers.

After a year, the prognosis for young individuals who were hospitalised for myocardial infarction is usually good. Nonetheless, early and vigorous care may be beneficial for a portion of these young patients, particularly those with a history of previous infarctions, a history of congestive heart failure, and the development of atrial fibrillation while receiving hospital treatment [59]. Despite more significant cardiac damage, younger patients experience lower mortality rates, fewer complications and favorable prognosis compared to older individuals [58]. Smoking plays an important role in Myocardial infarction occurrence in younger patients, while hypertension and diabetes are less of a concern for this group [60, 61].

CONCLUSION

In conclusion, while factors such as gender, family history and smoking have been associated with the risk of developing Myocardial infarction, younger individuals show distinct patterns in terms of disease progression and outcomes. The incidence and prevalence of MI will be considerably reduced

by increasing public knowledge of this problem, highlighting the importance of the "golden hour" and encouraging early identification and treatment.

REFERENCES

- Ojha N, Dhamoon AS. Myocardial Infarction. 2023; In StatPearls. StatPearls Publishing.
- Brscic E, Bergerone S, Gagnor A, et al. Acute myocardial infarction in young adults: prognostic role of angiotensinconverting enzyme, angiotensin II type I receptor, apolipoprotein E, endothelial constitutive nitric oxide synthase, and glycoprotein IIIa genetic polymorphisms at medium-term follow-up. Am Heart J. 2000; 139(6):979-84.
- Bhardwaj R, Kandoria A, Sharma R. Myocardial infarction in young adults-risk factors and pattern of coronary artery involvement. Niger J Med. 2014; 55(1):44-7. https://doi.org/10.4103/0300-1652.128161.
- Concheiro-Guisán A, Sousa-Rouco C, Fernández-Santamarina I, et al. Intrauterine myocardial infarction: unsuspected diagnosis in the delivery room. Fetal Pediatr Pathol. 2006; 25(4):179-84. https://doi.org/10.1080/15513810601015605.
- Reich JD, Campbell R. Myocardial infarction in children. Am J Emerg Med. 1998; 16(3):296-303. https://doi.org/10.1016/s0735-6757(98)90107-3.
- Celermajer DS, Sholler GF, Howman-Giles R, et al. Myocardial infarction in childhood: clinical analysis of 17 cases and medium term follow up of survivors. Heart. 1991; 65(6):332-6. https://doi.org/10.1136/hrt.65.6.332.
- 7. Gulati R, Behfar A, Narula J, *et al*. Acute Myocardial Infarction in Young Individuals. Mayo Clin Proc. 2020; 95(1):136–56. https://doi.org/10.1016/j.mayocp.2019.05.001.
- 8. Safdar B. Clues to Diagnose Myocardial Infarction in the Young.

 J Am Coll Cardiol. 2019; 73(5):585–
 8. https://doi.org/10.1016/j.jacc.2018.11.034.
- 9. Tresch DD, Brady WJ, Aufderheide TP, *et al.* Comparison of elderly and younger patients with out-of-hospital chest pain. Arch. Intern. Med. 1996; 156(10):1089-93.
- 10. Sagris M, Theofilis P, Mistakidou V, *et al.* Young and older patients with acute myocardial infarction: differences in risk factors and angiographic characteristics. *Hellenic journal of cardiology: HJC = Hellenike kardiologike epitheorese.* 2024; S1109-9666(24)00112-X.

https://doi.org/10.1016/j.hjc.2024.05.008

- 11. Lawesson SS, Stenestrand U, Lagerqvist B, *et al.* Gender perspective on risk factors, coronary lesions and long-term outcome in young patients with ST-elevation myocardial infarction. Heart. 2010; 96:453–459.
- 12. Alfaddagh A, Khraishah H, Rashed W, *et al.* Clinical characteristics and outcomes of young adults with first myocardial infarction: results from Gulf COAST. Int J Cardiol Heart Vasc. 2020; 31:100680.
- Gazit AZ, Avari JN, Balzer DT, et al. Electrocardiographic diagnosis of myocardial ischemia in children: is a diagnostic electrocardiogram always diagnostic?. Pediatrics. 2007; 120(2):440-4. https://doi.org/10.1542/peds.2007-0170.
- Reimer KA, Jennings RB, Tatum AH. Pathobiology of acute myocardial ischemia: metabolic, functional and ultrastructural studies. Am J Cardiol. 1983; 52(2):72-81. https://doi.org/10.1016/0002-9149(83)90180-7.
- 15. Manzar KJ, Padder FA, Conrad AR, et al. Acute myocardial

- infarction with normal coronary artery: a case report and review of literature. Am J Med Sci. 1997; 314(5):342-5.https://doi.org/10.1097/00000441-199711000-00013.
- 16. Vreede AP, Bockenstedt PL, Knight JS. Antiphospholipid syndrome: an update for clinicians and scientists. Curr. Opin. Rheumatol. 2017; 29(5):458-66.https://doi.org/10.1097/BOR.00000000000000410.
- Pericleous C, Ripoll VM, Giles I, et al. Laboratory tests for the antiphospholipid syndrome. Systemic Lupus Erythematosus: Methods Protoc. 2014:221-35.https://doi.org/10.1007/978-1-4939-0326-9 17.
- 18. Jouhikainen T, Pohjola-Sintonen S, Stephansson E. Lupus anticoagulant and cardiac manifestations in systemic lupus erythematosus. Lupus. 1994; 3(3):167-72. https://doi.org/10.1177/096120339400300307.
- 19. Ravelli A, Martini A. Antiphospholipid antibody syndrome in pediatric patients. *Rheum Dis Clin North Am.* 1997; 23(3):657-76.
- Osula S, Bell GM, Hornung RS. Acute myocardial infarction in young adults: causes and management. Postgrad. Med. J. 2002; 78(915):27-30.https://doi.org/10.1136/pmj.78.915.27.
- 21. Klues HG, Schwarz ER, vom Dahl J, *et al.* Disturbed intracoronary hemodynamics in myocardial bridging: early normalization by intracoronary stent placement: Early normalization by intracoronary Stent placement. 1997; 96(9), 2905–2913. https://doi.org/10.1161/01.cir.96.9.2905.
- 22. Wu WH, Sun JP, Ma L, *et al.* Anomalous origin of the left coronary artery from the pulmonary trunk. Int J Cardiol. 2015; 201:165–167. https://doi.org/10.1016/j.ijcard.2015.08.041.
- 23. Amaral ME, Epifânio P, Noronha N, *et al.* Unusual cause of left ventricular dysfunction in a child. Rev Port Cardiol. (English Edition). 2019; 38(2):159-e. https://doi.org/10.1016/j.repce.2017.07.015.
- 24. Farouk A, Zahka K, Siwik E, *et al.* Anomalous origin of the left coronary artery from the right pulmonary artery. J. Card. Sur. 2009; 24(1):49-54. https://doi.org/10.1111/j.1540-8191.2008.00622.x.
- 25. Ferreras C, Mota S, Sarmento JA, *et al.* ALCAPA syndrome: A rare etiology of cardiac arrest in a teenager. Int J Pediatr Adolesc Med. 2021; 8(4):271-2. https://doi.org/10.1016/j.ijpam.2021.04.001.
- 26. Taylor AJ, Rogan KM, Virmani R. Sudden cardiac death associated with isolated congenital coronary artery anomalies. J Am Coll Cardiol. 1992; 20(3):640-7. https://doi.org/10.1016/0735-1097(92)90019-j.
- 27. Yildiz A, Okcun B, Peker T, Arslan C, Olcay A, Bulent Vatan M. Prevalence of coronary artery anomalies in 12,457 adult patients who underwent coronary angiography. Clin Cardiol. 2010 33(12):E60-4. https://doi.org/10.1002/clc.20588.
- 28. Ghadri JR, Kazakauskaite E, Braunschweig S, *et al.* Congenital coronary anomalies detected by coronary computed tomography compared to invasive coronary angiography. BMC Cardiovascular Disorders. 2014;14:1-0. BMC cardiovasc. disord. 14(1). https://doi.org/10.1186/1471-2261-14-81.
- 29. Stout, K.K., Daniels, *et al.* 2019. 2018 AHA/ACC guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*, 73(12), pp.e81-e192. https://doi.org/10.1161/cir.000000000000000003.
- 30. Cepas-Guillén PL, Flores-Umanzor EJ, Sabate M, *et al.* Multivessel spontaneous coronary artery dissection involving the

- left main coronary artery: a case report. Eur. Heart J. Case Rep. 2019; 3(1):yty168.. https://doi.org/10.1093/ehjcr/yty168.
- 31. Vuorio AF, Kontula K, Turtola H, *et al.* Postmortem molecularly defined familial hypercholesterolemia and sudden cardiac death of young men. Forensic Sci. Int. 1999; 106(2):87-92. https://doi.org/10.1016/s0379-0738(99)00149-8.
- 32. Guthikonda S, Haynes WG. Homocysteine: role and implications in atherosclerosis. Curr. Atheroscler. Rep. 2006; 8(2):100-6.https://doi.org/10.1007/s11883-006-0046-4.
- 33. Zhang S, Bai YY, Luo LM, *et al.* Association between serum homocysteine and arterial stiffness in elderly: a community-based study. J Geriatr Cardiol. 2014; 11(1):32. https://doi.org/10.3969/j.issn.1671-5411.2014.01.007.
- 34. Stary HC. Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults. Arteriosclerosis (Dallas, Tex.). 1989; 9(1 Suppl):I19-32.
- 35. Nissen SE, Nicholls SJ, Sipahi I, *et al.* Effect of very high-intensity statin therapy on regression of coronary atherosclerosis: the ASTEROID trial. Jama. 2006; 295(13):1556-65. https://doi.org/10.1001/jama.295.13.jpc60002.
- 36. Strong JP, Malcom GT, McMahan CA, Tracy RE, Newman III WP, Herderick EE, Cornhill JF, Pathobiological Determinants of Atherosclerosis in Youth Research Group, Pathobiological Determinants of Atherosclerosis in Youth Research Group. Prevalence and extent of atherosclerosis in adolescents and young adults: implications for prevention from the Pathobiological Determinants of Atherosclerosis in Youth Study. Jama. 1999 Feb 24; 281(8):727-35. https://doi.org/10.1001/jama.281.8.727.
- 37. Mehta PM, Grainger TA, Lust RM, *et al*. Effect of cocaine on left ventricular function: relation to increased wall stress and persistence after treatment. Circulation. 1995; 91(12):3002-9. https://doi.org/10.1161/01.cir.91.12.3002.
- Mittleman MA, Mintzer D, Maclure M, et al. Triggering of myocardial infarction by cocaine. Circulation. 1999; 99(21):2737-41.https://doi.org/10.1161/01.cir.99.21.2737.
- 39. Taubert KA, Rowley AH, Shulman ST. Nationwide survey of Kawasaki disease and acute rheumatic fever. J. Pediatr. 1991; 119(2):279-82. https://doi.org/10.1016/s0022-3476(05)80742-5.
- 40. Kawasaki T, Kosaki F, Okawa S, *et al.* A new infantile acute febrile mucocutaneous lymph node syndrome (MLNS) prevailing in Japan. Pediatrics. 1974; 54(3):271-6.
- 41. Burns JC., Glodé MP. Kawasaki syndrome. Lancet. 364(9433), 533–544. https://doi.org/10.1016/S0140-6736(04)16814-1.]
- 42. Senzaki H. Long-term outcome of Kawasaki disease. *Circulation*. 2008;118(25):2763-
 - 72. https://doi.org/10.1161/CIRCULATIONAHA.107.749515.
- 43. Kato H, Sugimura T, Akagi T, *et al.* Long-term consequences of Kawasaki disease: a 10-to 21-year follow-up study of 594 patients. Circulation. 1996; 94(6):1379-85.https://doi.org/10.1161/01.cir.94.6.1379.
- 44. Brscic E, Bergerone S, Gagnor A, *et al.* Acute myocardial infarction in young adults: prognostic role of angiotensin-converting enzyme, angiotensin II type I receptor, apolipoprotein E, endothelial constitutive nitric oxide synthase, and glycoprotein IIIa genetic polymorphisms at medium-term follow-up. Am. Heart J. 2000; 139(6):979-84.https://doi.org/10.1067/mhj.2000.106165.
- 45. Egred M, Viswanathan G, Davis GK. Myocardial infarction in young adults. Postgrad. Med. J. 2005; 81(962):741-5. https://doi.org/10.1136/pgmj.2004.027532.
- 46. Mannucci PM, Asselta R, Duga S, *et al*. The association of factor V Leiden with myocardial infarction is replicated in 1880 patients

- with premature disease. J Thromb Haemost. 2010; 8(10):2116-21. https://doi.org/10.1111/j.1538-7836.2010.03982.x.
- 47. Lee KW, Lip GY. Acute coronary syndromes: Virchow's triad revisited. Blood Coagul Fibrinolysis. 2003; 14(7):605-25.14(7), 605-625. https://doi.org/10.1097/01.mbc.0000061355.73802.ea.
- Mostofsky E, van der Bom JG, Mukamal KJ, et al. Risk of myocardial infarction immediately after alcohol consumption. Epidemiology. 2015; 26(2):143-50. https://doi.org/10.1097/EDE.0000000000000227.
- 49. Solomon CG, Lee TH, Cook EF, *et al.* Comparison of clinical presentation of acute myocardial infarction in patients older than 65 years of age to younger patients: the Multicenter Chest Pain Study experience. Am. J. Card. 1989; 63(12):772-6. https://doi.org/10.1016/0002-9149(89)90040-4.
- 50. Chew S, Ng SC. Hormone replacement therapy (HRT) and ischaemic heart disease: getting to the heart of the matter. Singapore Med J. 2002; 43(1):41-4.
- 51. Hoit BD, Gilpin EA, Henning H, *et al.* Myocardial infarction in young patients: an analysis by age subsets. Circulation. 1986; 74(4):712-21. https://doi.org/10.1161/01.cir.74.4.712.
- 52. Lane, J. R., & Ben-Shachar, G. Myocardial infarction in healthy adolescents. Pediatrics, 2007; 120(4), e938–e943. https://doi.org/10.1542/peds.2006-3123
- Pellaton C, Monney P, Ludman AJ, et al. Clinical features of myocardial infarction and myocarditis in young adults: a retrospective study. BMJ open. 2012;2(6):e001571. https://doi.org/10.1136/bmjopen-2012-001571.
- 54. Holmes DR, White HD, Pieper KS, Ellis SG, Califf RM, Topol EJ. Effect of age on outcome with primary angioplasty versus thrombolysis. J Am Coll Cardiol. 1999; 33(2):412-9. https://doi.org/10.1016/s0735-1097(98)00579-8.
- 55. Holmes DR, White HD, Pieper KS, *et al*. Effect of age on outcome with primary angioplasty versus thrombolysis. J Am Coll Cardiol.

- 1999; 33(2):412-9. <a href="https://doi.org/10.1002/(sici)1097-0304(199811)45:3<280::aid-ccd14>3.0.co;2-p.">https://doi.org/10.1002/(sici)1097-0304(199811)45:3<280::aid-ccd14>3.0.co;2-p.
- Fahal IH, McClelland P, Hay CC, et al. Arterial thrombosis in the nephrotic syndrome. Postgrad. Med. J. 1994; 70(830):905-9.. https://doi.org/10.1136/pgmj.70.830.905
- 57. Henning H, Gilpin EA, Covell JW, *et al.* Prognosis after acute myocardial infarction: a multivariate analysis of mortality and survival. Circulation. 1979; 59(6):1124-36. https://doi.org/10.1161/01.cir.59.6.1124.
- Zukel WJ, Cohen BM, Mattingly TW, et al. Survival following first diagnosis of coronary heart disease. Am. Heart J. 1969; 78(2):159-70.https://doi.org/10.1016/0002-8703(69)90004-0.
- 59. Lytle BW, Kramer JR, Golding LR, *et al.* Young adults with coronary atherosclerosis: 10 year results of surgical myocardial revascularization. Am Coll Cardiol. 1984; 4(3):445-53. https://doi.org/10.1016/s0735-1097(84)80086-8.
- 60. Kennelly BM. Aetiology and risk factors in young patients with recent acute myocardial infarction. S. Afr. Med. J. 1982; 61(14):503-7.
- 61. Rissanen AM. Familial occurrence of coronary heart disease: effect of age at diagnosis. Am. J. Card. 1979; 44(1):60-6. https://doi.org/10.1016/0002-9149(79)90251-0

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