Journal of Cardiothoracic Surgery

CASE REPORT

Open Access

Complete obstruction of proximal left ascending artery in a very young woman with Kawasaki: a case report and literature review

Pouya Ebrahimi[1], Mohammad Hossein Mandegar[2], Amir Nasrollahizadeh[1], Parnian Soltani[1], Hamidreza Soleimani[3], Pedram Ramezani[4] and Fatemeh Naderi[5*]

Abstract

Introduction Kawasaki disease is a predisposing factor for various potentially fatal anomalies that can cause coronary artery diseases in childhood, adolescence, or adulthood if they are not appropriately treated, usually done with ASA and intravenous immunoglobulin (IVIG). This study presents a case of a young woman who needed a coronary artery bypass graft (CABG) due to a history of Kawasaki and coronary artery severe stenosis.

Case presentation A 24-year-old woman presented with chest pain, exertional and at-rest dyspnea, lower limb edema, and crackle at bilateral lung bases. After stabilization, it was noted that her left anterior descending artery (LAD) had been obstructed completely. She underwent CABG successfully and recovered uneventfully. The six-month follow-up showed almost complete. His medical therapy continued with dual antiplatelet therapy, statin, diuretics, spironolactone, and some other medications to decline the process of her heart structural change (Graphical Abstract).

Results Kawasaki makes the patients prone to CAD and several other CVD diseases. Many factors determine the risk of coronary artery involvement in these cases, and the proffered treatment should be chosen based on the severity of CAD and the characteristics of the patient. These treatments include (1. Medical, 2. Interventional (PCI), 3. Surgical (CABG), 4. Combinational) along with preventive therapy, which is suggested to almost all patients. In our case, considering the complete occlusion of LAD, the patient underwent CABG.

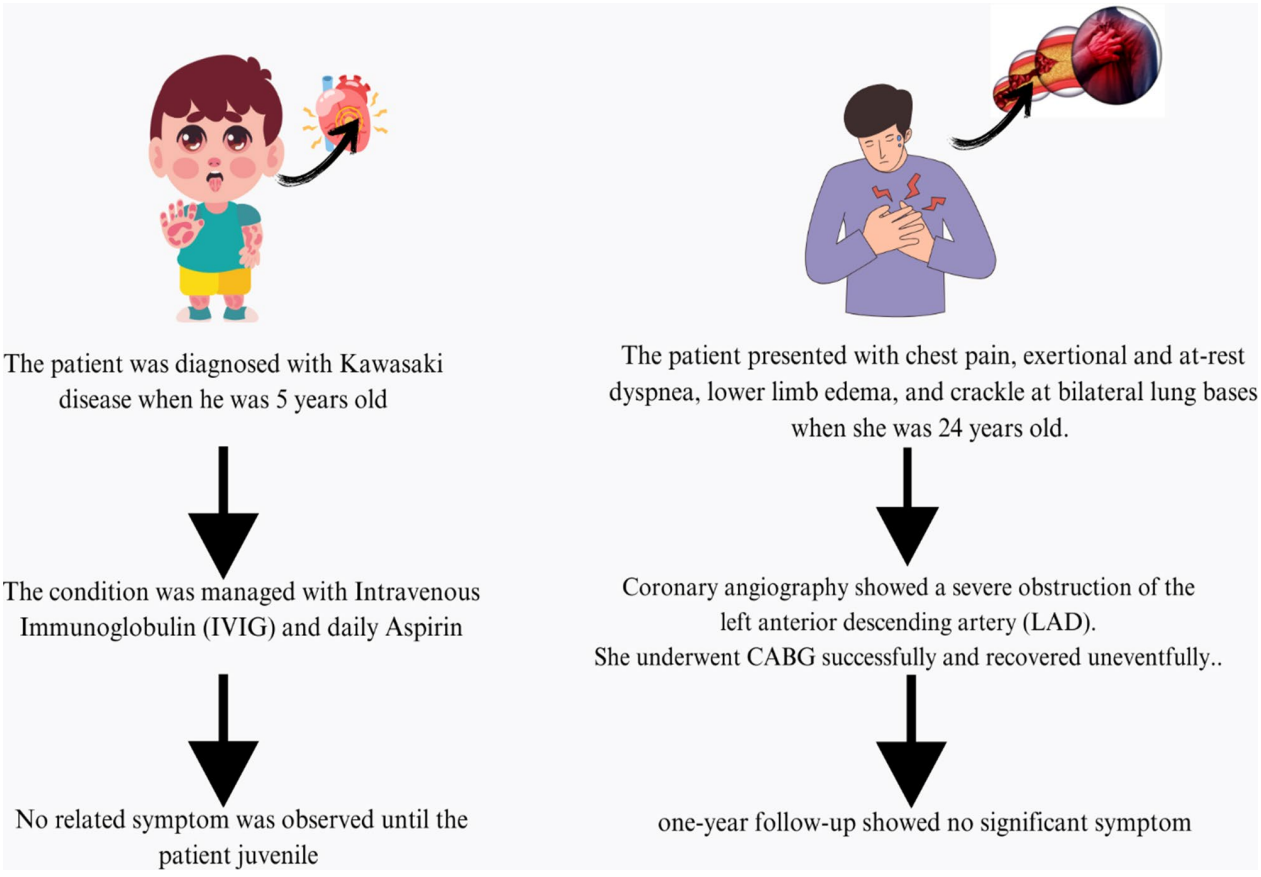
Clinical key point Kawasaki disease is one of the most important predisposing factors for CAD in young adults, and their least significant symptoms should be taken seriously as a CVD and should be investigated. CAD in these patients might be so severe that no treatment except CABG can be responsive to problem-solving. Moreover, timely management of disease in childhood with ASA and IVIG would prevent or at least decrease the severity of symptoms can be declined significantly.

Keywords Kawasaki disease, Ischemic heart disease, Heart failure, Case report

*Correspondence:
Fatemeh Naderi
fatemeh_naderi2010@yahoo.com

Full list of author information is available at the end of the article

Graphical Abstract



Introduction

Kawasaki disease is a systemic and usually self-limited mucocutaneous vasculitis syndrome that generally involves lymph nodes and is mainly seen in infants and children. This disease consists of the vasculitis system all over the body. Its etiology has yet to be known, and its prevalence (diagnosis) has increased since its introduction in 1967 [1, 2]. Several health conditions, such as coronary artery aneurysm (CAA), accompany this disease. Therefore, it is considered a grave disease with 30–50% progression to CA ectasia or progression if untreated [3, 4]. Although most children are now rarely diagnosed and treated with ASA and IVIG, up to 50% of patients are irresponsive, and they are more prone to CAD in childhood or early adulthood and even sudden cardiac death [3, 4]. It has been shown in a previous study that almost 5% of young patients with ACS might have been affected by antecedent Kawasaki disease [5].

The main arteries involved in Kawasaki are medium-sized arteries, including coronary arteries, mostly in the form of unreversible CAA [6]. These aneurysms can be considerable life-long risk factors for fatal thromboembolic events or myocardial infarctions in the next years of lives [7, 8]. These conditions are triggered by the turbulence of blood flow in cavernous CAAs, which can trigger thrombosis formation. In contrast, atherosclerotic changes (cholesterol crystals, macrophage infiltrations, and thrombosis) could be seen in both CAAs and other parts of the coronary arteries when investigated by OTC, which provides visual insight from the inside of the vessels [8–10]. If this condition progresses further, it can result in infarction of the ventricular muscle and reduced LVEF, which finally presents as DHF symptoms like the presented symptoms in the presented patient (dyspnea, edema of lower extremities, crackle, and lung auscultation) [11].

In this study, we present a young woman with symptoms and signs of CAD and HF due to the sequels that had been unaddressed on coronary arteries during her childhood.

Case presentation

The patient was a 24-year-old woman who presented with an intermittent Shortness of breath (SOB) and declared a history of Kawasaki disease in her infancy. The symptoms were not severe and had not affected the daily

activities significantly. On her physical exam, no abnormal finding was observed, and vital signs were stable. Considering her past medical history and background with Kawasaki disease, cardiovascular diseases were suggested as a responsible factor. The background disease was diagnosed with ASA and IVIG and had no other manifestations after her childhood. No past FH or PMH was declared. No history of smoking, previous surgery, or allergy to food and medications was declared.

Therefore, a CT angiography was planned, which showed a mild narrowing of LAD, which was not significant, and patency of other coronary vessels (Fig. 1). The patient was discharged with a recommendation to take ASA 80 mg daily, Clopidogrel 75 mg daily, and Atorvastatin 40 mg once a night. In her follow-up, no significant change was seen. However, after ten years, when she was 34, she presented with overt symptoms of chest pain at exertion and rest and exertional SOB, suggestive of CAD and DHF. On general appearance, an anxious woman with evident dyspnea could be observed. On vital sign evaluation, the oxygen saturation on ambient air declined (86%), normal blood pressure (130/90), high respiratory rate [28], and tachycardia (111) could be observed. The physical examination showed bilateral lung crackles and 2 + edema was seen on the lower extremities. The respiration was performed with the help of accessory abdominal muscles.

Methods

Considering the critical condition, the patient was admitted to the ED, primary therapeutic interventions such as nasal via Oxygen cannula, ASA (300 mg stat orally), Clopidogrel (325 mg orally), Furosemide (80 mg), Atorvastatin (40 mg), IV UFH (5000 IU), Metoprolol (50 mg orally), Morphine Sulphate (3 mg), and IV Nitroglycerin (5 micrograms per minute) was initiated immediately. A semi-sitting position was applied, and a Foley catheter was inserted to check the patient's intake and output. The patient's condition improved significantly after the primary therapeutic interventions. Simultaneously, ECG and bedside TTE were performed. The ECG showed ischemic changes (Inverted T-wave and ST-segment changes). TTE showed

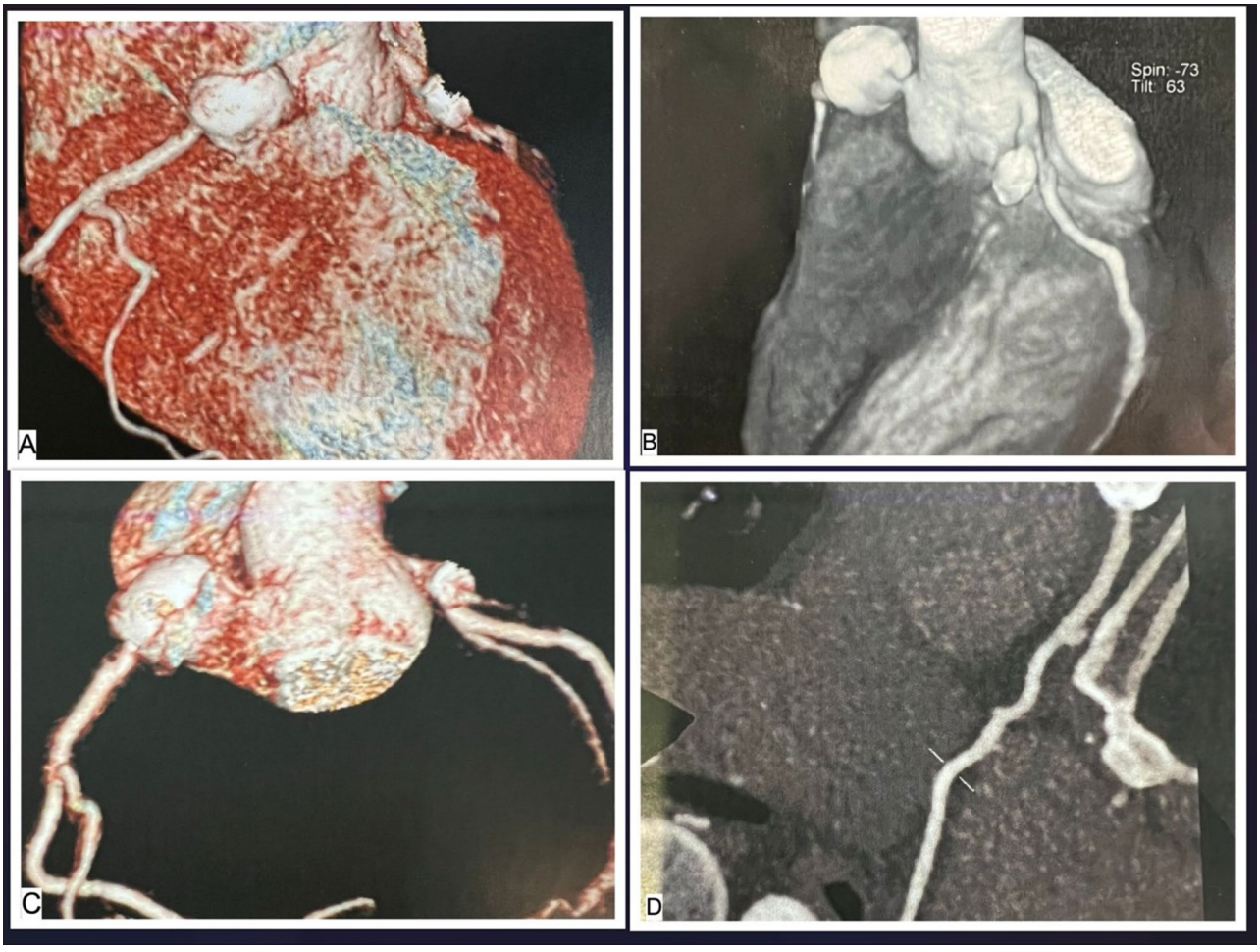


Fig. 1 The initial evaluation at her age of 24 showed mild stenosis of the Left anterior descending artery and patency of other coronary arteries

a decline in LVEF (25–30%). The clinical condition was improved, the SOB declined, and the crackle of the lungs and edema improved remarkably. After stabilization, the patient was stabilized and transferred to the CCU. The day after that, the patient was prepared to undergo diagnostic CAG (Fig. 2). The result showed a significant almost complete obstruction of the proximal LAD. The severe calcified obstruction was deemed so severe and unlikely to be adequately predilated with a balloon that it was impossible to resolve with percutaneous coronary intervention (PCI) (Fig. 2); therefore, due to nearly completed obstruction of LAD with findings of large anterior ischemia, the heart team planned a coronary bypass artery graft (CABG). Laboratory data, including High-sensitivity troponin and creatinine kinase-MB, was normal.

The CABG was performed on the same day, and a graft was used to replace the LAD vessel without specific complications. The stricture was resolved, and post-surgery CAG showed improved blood flow to the ventricular muscle and LV function (LVEF:35–40%). The treatment with DAPT (ASA + Plavix), Statins, Beta-blockers, ACEI, Diuretics (furosemide), and regular clinical and TTE follow-ups was planned. A 6-month follow-up of the patient showed a significantly improved medical condition and TTE parameter, suggestive of response to surgery and medications. At one year follow-up, the same favorable results with no significant new symptoms and improved quality of life were claimed. According to the AHA statement, the follow-up plan including obtaining ECG and echocardiography every six months, and invasive coronary angiography if indicated was planned [12].

Discussion

Kawasaki-induced CVD, and especially MI and HF, have been shown to be not commonly reported complications of childhood Kawasaki, especially in those who had not received necessary treatments during the vasculitis in medium-sized vessels. CAAs, which are caused by this condition, are a predisposing factor for catastrophic cardiac events such as MI and SCD caused by resultant thrombosis. The most common occurrence of these potentially lethal conditions is within 2 years after the onset of Kawasaki

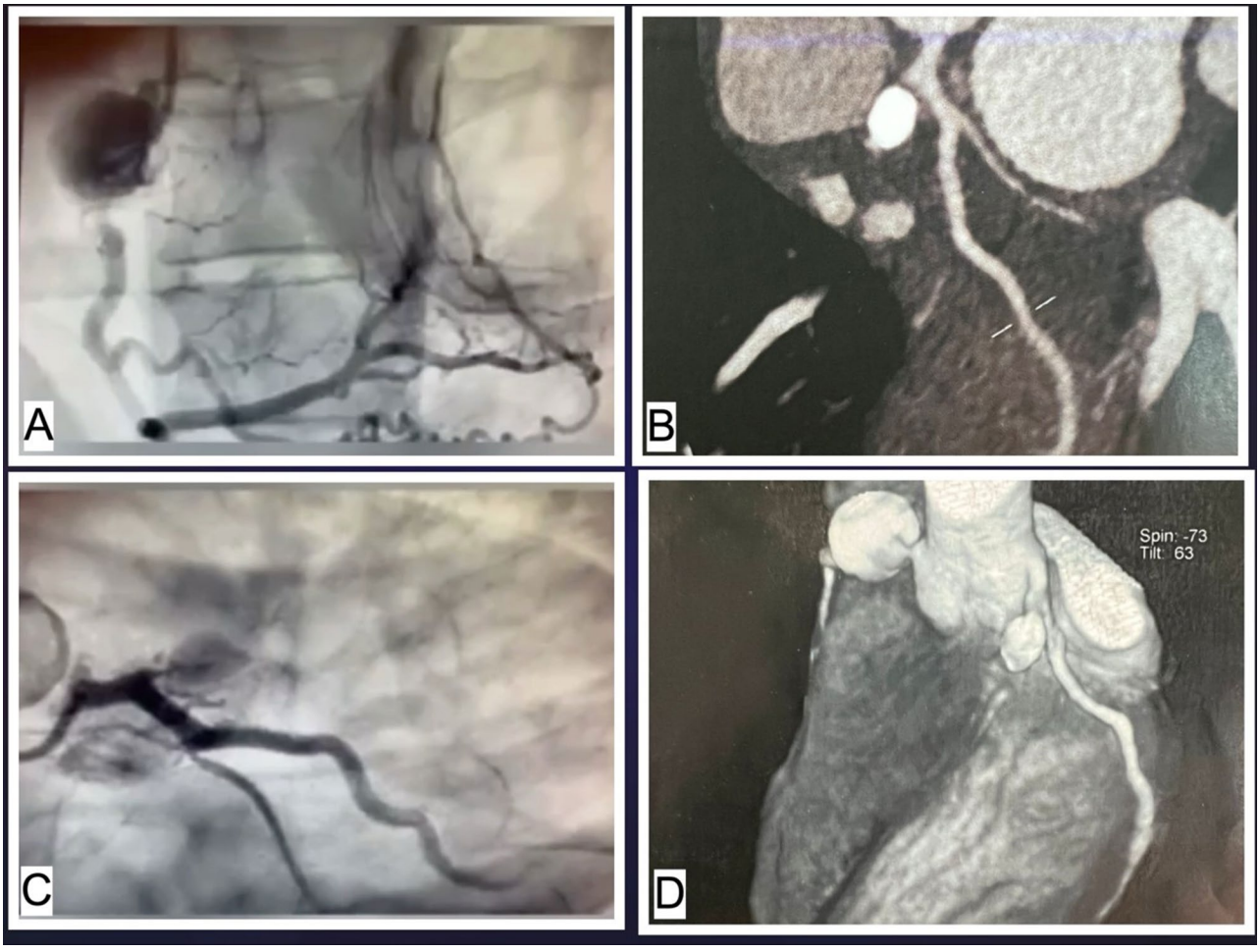


Fig. 2 Severe stenosis of the Left Ascending Descending Artery (LAD) could be detected on Coronary angiography (CAG)

disease [13, 14]. In this report, we presented a case of a young woman who needed an aggressive CABG surgery due to the severity of one of the main coronaries (LAD) involvement. It underscores the higher risk of incidence of severe CVD in these patients, even years after their childhood [11].

The long-term complications of Kawasaki Syndrome have been categorized into several categories (1. coronary artery abnormalities, 2. subclinical vascular outcomes, 3. valvular regurgitation, 4. aortic regurgitation, 5. myocardial abnormalities, 6. arrhythmias), and a risk stratification system has been introduced that can predict the complications (Table 1) [15].

Based on the AHA/ACC guidelines, other risk factors play a role in determining these patients' coronary involvement risk [15]. These factors include: (1) Greater length and distal location of aneurysms◊ increased flow stasis, (2) Increased aneurysm number, (3) Increased involved branches number, (4) luminal irregularities presence, (5) Abnormal vessel wall feature (calcification, luminal myoblast feature change), (6) impaired vasodilation, impaired flow reserve, (7) Poor luminal vessel quality r presence, (8) Presence of previous MI, or VD, revascularization, or thrombosis [15].

Several possible predisposing factors have been proposed regarding CA involvement and increased risk of CAD in Kawasaki patients, including inflammatory process, endothelial dysfunction, intima thickening, increased matrix metalloproteinases, and vascular damage and interruption of blood flow [16, 17].

Table 1 Risk classification of coronary artery abnormalities during Follow-up [15]

Classification		Description
1		No involvement at any time point (Z score always < 2)
2		Dilation only (Z score 2 to < 2.5)
3	3	Small aneurysm (Z score ≥ 2.5 to < 5)
	3.1	Current or persistent
	3.2	Decreased to dilation only or normal luminal dimension
4	4	Medium aneurysm (Z score ≥ 5 to < 10, and absolute dimension < 8 mm)
	4.1	Current or persistent
	4.2	Decreased to small aneurysm
	4.3	Decreased to dilation only or normal luminal dimension
5	5	Large and giant aneurysm (Z score ≥ 10, or absolute dimension ≥ 8 mm)
	5.1	Current or persistent
	5.2	Decreased to medium aneurysm
	5.3	Decreased to small aneurysm
	5.4	Decreased to dilation only or normal luminal dimension

*The Z-score is calculated based on the luminal diameter by TEE, and then it was modified by calculating it based on the Z-score/BSA

Regarding pharmaceutical management of Kawasaki disease, recent studies declare the noninferiority of medium-dose aspirin vs. high-dose aspirin for improved CA outcomes [18]. However, in cases of CAD and significant thrombosis, a combination therapy of antiplatelet and anticoagulant drugs is preferred [15]. Statin usage has been shown to reduce inflammation and improve endothelial cell function in Kawasaki patients besides their protective effects in secondary CVD prevention [19]. Furthermore, in cases of DHF as our patient, diuretics were prescribed to mitigate the volume overload [20].

Sometimes the extent of coronary artery involvement is severe and mechanical revascularization is needed: 1) Adults with KD history who are diagnosed with STEMI (should be referred for emergency angiography to determine the type of revascularization, 2) Adult KD + NSTMI + high-risk CAD ◊(multivessel + declined LVEF, LMA, multivessel + DM, NTEMI + anatomy appropriate for revascularization on PCI on CAG, UA + non-responsive for medical therapy, CAD involing ≥ 10% LV muscle mass) [15].

Once the need for revascularization is determined, choosing between interventional (PCI) and surgical (CABG) methods is time. Several factors are in favor of performing CABG, such as:

1. Presence of ≥ 1 chronic total occlusion.
2. Patients with multivessel coronary artery involvement and reduced LV function (because of either prior MI or chronic ischemia).
3. Diabetic patients.

Considering the complete occlusion of the LMA, our patient fulfills the first criteria. Therefore, she was considered for CABG and was successfully managed without any significant complications.

Moreover, several studies declared the superiority of CABG compared to PCI, mostly due to lower rates of need for revascularization and more favorable outcomes in complex cases and multivessel involvement [21, 22].

CABG is specifically indicated for young patients with severe coronary complications, ensuring long-term patency and survival benefits. It has been revealed that arterial grafts, such as the mammary arteries, are preferred due to their better durability and patency than venous grafts. Long-term follow-ups have shown patency rates of more than 82%, with mortality rates remaining low, thus proving the effectiveness of CABG in this regard [23]. Mixed grafting strategies further improve the outcomes by playing to the strengths of both arterial and venous conduits [24].We conducted a comprehensive search protocol with the following search strategy ("Kawasaki disease"[Mesh] OR "Kawasaki syndrome"[tiab] OR "mucocutaneous lymph node syndrome"[tiab]) AND ("Coronary artery bypass"[Mesh] OR "CABG"[tiab] OR "coronary

Table 2 Similar cases of young-aged coronary artery diseases need coronary artery bypass graft

Title	Number of patients	Demographics/Medical History	Culprit Vessel	Main Findings/Outcomes
Robotic Minimally Invasive Direct Coronary Artery Bypass for Kawasaki Disease Matsumoto et al. 2020[25]	3	Young aged patients (16 to 30 years old) with KD diagnosis in early childhood	LAD	Proximal LAD aneurysms and stenosis were successfully treated by DaVinci surgical system-assisted of-pump CABG. Excellent symptom relief.
Unexpected Evolution After Multivessel Coronary Artery Bypass Grafting in a Patient with Kawasaki Disease Victor et al. 2021[26]	1	A 17-year-old Caucasian boy presented with chest pain	LAD and RCA	Right and left internal mammary arteries were utilized to bypass the RCA and LAD, but the patient had a complicated medical course following the surgery with several NSTEMI which needed repeated interventions.
Off-pump coronary bypass grafting for Kawasaki disease Verma S. et al. 2010 [27]	1	6-year-old girl; history of acute KD and thrombotic occlusion	RCA	Successful off-pump CABG with right internal mammary arteries graft; good postoperative recovery and graft patency.
Coronary artery bypass graft in Kawasaki disease patients: Siriraj experience Chanthong P. et al. 2005 [28]	5	Ages: 3.3–14.4 years; only 2 patients presented with chest pain, delayed diagnosis in some cases	LAD, RCA, LCx and LM	All patients had successful CABG with no perioperative deaths; postoperatively all of them had NYHA I.
Coronary artery bypass grafting for Kawasaki disease Guo H.W. et al. 2010 [29]	8	Ages: 4–40 years; history of significant coronary anomalies	LAD, RCA, and LM	CABG improved symptoms; long-term efficacy needs further follow-up.
Long-term clinical outcomes of coronary artery bypass grafting in young children with Kawasaki disease Kwak Y. et al. 2022 [30]	6	Median age at CABG: 13 years; all male	LAD and RCA	All grafts patent; postoperative improvements in myocardial perfusion and no operative complications.
Coronary artery bypass grafting for Kawasaki disease Gotteiner et al. (2002) [31]	5	Pediatric cases (from 8 months to 12 years old) with significant coronary complications	LAD and RCA	CABG is noted as safe and effective for the long-term of severe coronary lesions in KD patients resulting in improvement of NYHA to class I, postoperatively.
Coronary artery bypass grafting in a child with Kawasaki disease Magro et al. (2021) [32]	1	23-month-old boy; late diagnosis of KD; large coronary aneurysms	LAD	Successful left internal mammary artery to LAD graft; no complications and good long-term graft patency.

Left Ascending Descending Artery (LAD), Right Coronary Artery (RCA), Left Circumflex artery (LCx), Left Main (LM), Coronary Artery Bypass Graft (CABG), Kawasaki Disease (KD), New York Heart Association Class (NYHA), Non-ST-elevation myocardial infarction

bypass"[tiab] OR "coronary artery surgery"[tiab] OR "myocardial revascularization"[Mesh]) AND ("1980/01/01"[Date - Publication]: "2024/12/31"[Date - Publication]) in PubMed database. Table 2 represents the most detailed and educational cases of CABG in Kawasaki patients.

Clinical key point: Kawasaki disease is one of the most important predisposing factors for CAD in young adults, and their least significant symptoms should be taken seriously as a CVD and should be investigated. CAD in these patients might be so severe that no treatment except CABG can be responsive to problem-solving. Moreover, timely management of disease in childhood with ASA and IVIG would prevent or at least decrease the severity of symptoms can be declined significantly.

Abbreviations

ACEI	Angiotensinogen Receptor Converting Enzyme Inhibitors
BSA	Body Surface Area
CAA	Coronary Artery Aneurysm
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CT	Computed Tomography
CVD	Cardiovascular Disease
DAPT	Dual Antiplatelet Therapy
DHF	Decompensated Heart Failure
ECG	Electrocardiogram
ED	Emergency Department
IV	Intravenous
IVIG	Intravenous Immunoglobulin
KD	Kawasaki Disease
LAD	Left Anterior Descending Artery
LV(EF)	Left Ventricle (Ejection Fraction)
OTC	Optical Coherence Tomography
PCI	Percutaneous Coronary Intervention
SOB	Shortness of Breath
TTE	Trans-Thoracic Echocardiography
UA	Unstable Angina
UFH	Unfractionated Heparin

Acknowledgements
None.

Author contributions
P.E., M.H.M., A.N., H.S., and P.S. contributed to data curation, supervision, project administration, data collection, analysis, supervision, writing the initial draft, and revision of the final manuscript script. P.R. and F.N. contributed to data collection, analysis, data curation, analysis of data, writing the initial draft, and revision of the final manuscript. All authors have read and approved the final version of the manuscript.

Funding
No funds were received for this study.

Data availability
Data is available upon reasonable request due to privacy/ethical restrictions.

Declarations

Ethical approval
Not applicable.

Consent to participate
The patient provided written informed consent to participate in this clinical case report, ensuring that all personal information and medical data will be kept confidential and used solely for research purposes.

Consent for publication
The patient provided informed consent for the publication of this report, and the center's ethical policy performed the procedure.

Competing interests
The authors declare no competing interests.

Author details
[1]Cardiovascular Diseases Research Institute, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran
[2]Department of Cardiovascular Surgery, Tehran University of Medical Sciences, Tehran, Iran
[3]Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[4]Non-Communicable Diseases Research Center, Endocrinology and Metabolism Population Sciences Institute, Tehran University of Medical Sciences, Tehran, Iran
[5]Pediatric Cardiovascular Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan 8174673461, Iran

Received: 22 November 2024 / Accepted: 11 April 2025
Published online: 23 April 2025

References

1. Uehara R, Belay ED. Epidemiology of Kawasaki disease in Asia, Europe, and the united States. J Epidemiol. 2012;22(2):79–85.
2. Chen S, Dong Y, Kiuchi MG, Wang J, Li R, Ling Z, et al. Coronary artery complication in Kawasaki disease and the importance of early intervention. JAMA Pediatr. 2016;170(12):1156.
3. Newburger JW, Takahashi M, Gerber MA, Gewitz MH, Tani LY, Burns JC, et al. Diagnosis, treatment, and Long-Term management of Kawasaki disease. Circulation. 2004;110(17):2747–71.
4. Daniels LB, Tjajadi MS, Walford HH, Jimenez-Fernandez S, Trofimenko V, Fick DB, et al. Prevalence of Kawasaki disease in young adults with suspected myocardial ischemia. Circulation. 2012;125(20):2447–53.
5. Shyu TC, Wu CJ, Fu YC, Peng YC, Chuang TY, Kuo HC et al. Prevalence of antecedent Kawasaki disease in young adults with suspected acute coronary syndrome in high incidence cohort. Front Cardiovasc Med. 2023;10.
6. Shiono Y, Takahata M, Ino Y, Tanimoto T, Kakimoto N, Suenaga T, et al. Pathological alterations of coronary arteries late after Kawasaki disease. JACC: Adv. 2024;3(6):100937.
7. Friedman KG, Gauvreau K, Hamaoka-Okamoto A, Tang A, Berry E, Tremoulet AH et al. Coronary artery aneurysms in Kawasaki disease: risk factors for progressive disease and adverse cardiac events in the US population. J Am Heart Assoc. 2016;5(9).
8. Kato T, Miura M, Kobayashi T, Kaneko T, Fukushima N, Suda K et al. Analysis of coronary arterial aneurysm regression in patients with Kawasaki disease by aneurysm severity: factors associated with regression. J Am Heart Assoc. 2023;12(3).
9. Tearney GJ, Regar E, Akasaka T, Adriaenssens T, Barlis P, Bezerra HG, et al. Consensus standards for acquisition, measurement, and reporting of intravascular optical coherence tomography studies. J Am Coll Cardiol. 2012;59(12):1058–72.
10. Takahashi K, Oharaseki T, Naoe S. Pathological study of postcoronary arteritis in adolescents and young adults: with reference to the relationship between sequelae of Kawasaki disease and atherosclerosis. Pediatr Cardiol. 2001;22(2):138–42.
11. Pilania RK, Jindal AK, Bhattarai D, Naganur SH, Singh S. Cardiovascular involvement in Kawasaki disease is much more than Mere coronary arteritis. Front Pediatr. 2020;8.
12. Manlhiot C, Niedra E, McCrindle BW. Long-term management of Kawasaki disease: implications for the adult patient. Pediatr Neonatol. 2013;54(1):12–21. https://doi.org/10.1016/j.pedneo.2012.12.013.
13. Fukazawa R, Kobayashi T, Mikami M, Saji T, Hamaoka K, Kato H, et al. Nationwide survey of patients with giant coronary aneurysm secondary to Kawasaki disease 1999–2010 in Japan. Circ J. 2018;82(1):239–46.
14. Atsuko S, Sachiko MT, Makoto N, Chikao Y. Remodeling of coronary artery lesions due to Kawasaki disease. Jpn Heart J. 2000;41(3):245–56.
15. McCrindle BW, Rowley AH, Newburger JW, Burns JC, Bolger AF, Gewitz M et al. Diagnosis, treatment, and Long-Term management of Kawasaki disease: A scientific statement for health professionals from the American heart association. Circulation. 2017;135(17).
16. Senzaki H. The pathophysiology of coronary artery aneurysms in Kawasaki disease: role of matrix metalloproteinases. Arch Dis Child. 2006;91(10):847–51. https://doi.org/10.1136/adc.2005.087437.
17. Zhang D, Liu L, Huang X, Tian J. Insights into coronary artery lesions in Kawasaki disease. Front Pediatr. 2020;8:493. https://doi.org/10.3389/fped.2020.00493. Published 2020 Aug 25.
18. Suzuki T, Michihata N, Hashimoto Y, et al. Association between aspirin dose and outcomes in patients with acute Kawasaki disease: a nationwide retrospective cohort study in Japan. Eur J Pediatr. 2024;183(1):415–24. https://doi.org/10.1007/s00431-023-05302-8.
19. Shimizu C, Kim J, He M, et al. RNA sequencing reveals beneficial effects of Atorvastatin on endothelial cells in acute Kawasaki disease. J Am Heart Assoc. 2022;11(14):e025408. https://doi.org/10.1161/JAHA.122.025408.
20. Bonnet M, Champagnac A, Lantelme P, Harbaoui B. Endomyocardial biopsy findings in Kawasaki-like disease associated with SARS-CoV-2. Eur Heart J. 2020;41(39):3863–4. https://doi.org/10.1093/eurheartj/ehaa588.
21. Muta H, Ishii M. Percutaneous coronary intervention versus coronary artery bypass grafting for stenotic lesions after Kawasaki disease. J Pediatr. 2010;157(1):120–6. https://doi.org/10.1016/j.jpeds.2010.01.032.
22. Dionne A, Bakloul M, Manlhiot C, et al. Coronary artery bypass grafting and percutaneous coronary intervention after Kawasaki disease: the pediatric Canadian series. Pediatr Cardiol. 2017;38(1):36–43. https://doi.org/10.1007/s00246-016-1480-x.
23. Yuan SM. Cardiac surgical procedures for the coronary sequelae of Kawasaki disease. Libyan J Med. 2012;7(1):19796.
24. Salsano A, Liao J, Miette A, Capoccia M, Mariscalco G, Santini F, et al. Surgical myocardial revascularization outcomes in Kawasaki disease: systematic review and meta-analysis. Open Med. 2021;16(1):375–86.
25. Matsumoto Y, Fukushima S, Shimahara Y, Kawamoto N, Tadokoro N, Kitamura S, et al. Robotic minimally invasive direct coronary artery bypass for Kawasaki disease. Gen Thorac Cardiovasc Surg. 2020;68(9):1037–9.
26. Victor S, McKillion KC, Puette JA, McKillion P, Ellison MB. Unexpected Evolution After Multivessel Coronary Artery Bypass Grafting in a Patient With Kawasaki Disease. Cureus. 2021.
27. Verma S, Dasarathan C, Premsekar R, Vaijyanath P. Off-pump coronary bypass grafting for Kawasaki disease. Ann Pediatr Cardiol. 2010;3(2):190.
28. Chanthong P, Sriyoschati S, Durongpisitkul K, Soongswang J, Laohaprasitiporn D, Nana A. Coronary artery bypass graft in Kawasaki disease patients: Siriraj experience. J Med Assoc Thai. 2005;88(Suppl 8):S197–202.
29. Guo Hwei, Chang Q, Xu J, ping, Song Y hu, Sun H. song, Hu S shou. Coronary artery bypass grafting for Kawasaki disease. Chin Med J (Engl). 2010;123(12):1533–6.
30. Kwak Y, Kwak JG, Cho S, Kim WH. Long-term clinical outcomes of coronary artery bypass grafting in young children with Kawasaki disease. Cardiol Young. 2022;32(3):459–64.
31. Gotteiner N, Mavroudis C, Backer CL, Pahl E, Duffy CE, Dalzell V, et al. Coronary artery bypass grafting for Kawasaki disease. Pediatr Cardiol. 2002;23(1):62–7.
32. Magro P, Carvalho N, Anjos R, Neves J. Coronary artery bypass grafting in a child with Kawasaki disease. Revista Portuguesa de Cardiologia (English Edition). 2021;40(7):519.e1-519.e4.

Publisher's note