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Acute Coronary Syndrome in Kawasaki Disease From Infancy to Adulthood

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ABSTRACT: Acute coronary syndrome (ACS) remains underrecognized in patients with Kawasaki disease (KD). The population at risk for KD-associated ACS is increasing, yet current ACS guidelines are largely derived from atherosclerotic disease and do not adequately address the distinctive mechanisms, anatomy, and thrombosis risk associated with KD. Because the pathophysiology and management of KD-associated ACS differ in fundamental ways from those of atherosclerotic coronary artery disease, its management requires a coordinated team response, with collaboration between pediatric and adult cardiologists. Recent advances in coronary imaging, interventional techniques, and antithrombotic therapies provide an opportunity to establish a more disease-specific approach. This review synthesizes current evidence on the epidemiology and pathophysiology of coronary artery aneurysms, interventional coronary imaging, acute management, revascularization strategies, antithrombotic therapy, and post-ACS surveillance in patients with KD to guide clinical decision-making and treatment for patients with KD presenting with ACS.

Key Words: acute coronary syndrome ■ coronary aneurysm ■ mucocutaneous lymph node syndrome ■ myocardial infarction ■ percutaneous coronary intervention

Kawasaki disease (KD) is an acute vasculitis that targets medium-sized muscular arteries. Its most serious long-term complication is the development of coronary artery aneurysms (CAAs).[1] Although early treatment has reduced the incidence of CAAs, a subset of patients develops persistent or remodeled coronary lesions, leading to lifelong risk for myocardial ischemia and myocardial infarction (MI).[2] Adults may also sometimes present with significant outward remodeling in the coronary arteries secondary to atherosclerotic disease with aneurysm and formation, yet the risk profile and treatment of such patients differs substantially from those in adult patients with KD with CAAs. Acute MI has emerged as a major cause of morbidity and mortality in pediatric and adult patients with KD.[3] Acute coronary syndrome (ACS) is defined as reduced blood flow to the coronary myocardium manifesting as ST-segment–elevation MI or non–ST-segment–elevation ACS, which includes unstable angina and non–ST-segment–elevation MI.[4] Because the pathophysiology and management of KD-associated ACS differ fundamentally from those of atherosclerotic coronary artery disease (ASCAD), timely recognition and a coordinated team response between pediatric and adult cardiologists are essential. Current ACS guidelines, largely based on atherosclerotic disease, do not adequately address the distinctive anatomy, mechanisms, and thrombosis risks specific to KD. This review synthesizes current evidence on the epidemiology, pathophysiology, and management of KD-associated ACS.

EPIDEMIOLOGY OF ACS IN PEDIATRIC AND ADULT PATIENTS WITH KD

In the modern era of intravenous immunoglobulin treatment, the incidence of persistent CAA 1 month after the onset of KD is 2.3% in Japan.[5] However, ACS remains a concern,

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Nonstandard Abbreviations and Acronyms

ACS	acute coronary syndrome
ASCAD	atherosclerotic coronary artery disease
CAA	coronary artery aneurysm
CABG	coronary artery bypass grafting
CT	computed tomography
CTO	chronic total occlusion
DAPT	dual antiplatelet therapy
KD	Kawasaki disease
KDCAS	coronary artery sequelae of Kawasaki disease
LVEF	left ventricular ejection fraction
MI	myocardial infarction
PCI	percutaneous coronary intervention

occurring almost exclusively in those who developed giant CAA with a Z score ≥10 or ≥8 mm during the acute phase.[6–10] Risk increases significantly in those with CA Z scores ≥20 or with a greater number of affected coronary branches.[6] Chronic ischemia causing heart failure and death has been reported in at least 1 patient with medium-sized aneurysms and involvement of multiple coronary arteries.[6] In a retrospective cohort of 1006 patients, major adverse coronary events occurred in 7 of 425 patients (2%) with medium-sized CAAs, demonstrating that this subgroup remains at risk for clinically significant coronary complications despite generally favorable long-term outcomes.[9]

In the pediatric population, the highest risk of ACS is concentrated within the first 2 years after KD onset.[6,9,10] One study of 209 patients with giant CAAs reported 10-year event-free survival of 68%.[10] Within the first 2 years, early deaths were primarily driven by MI or CAA rupture. North American cohorts mirror these findings, with adverse events over 10 years occurring only in children with Z scores ≥10.[6]

For adult survivors from childhood KD, Japanese registry data indicate that ACS accounts for 19.7% of hospitalizations for late KD sequelae.[11] These patients had a median age of 39 years and were predominantly male. Unlike patients with ASCAD, they often presented with a normal body mass index, although nearly 38% were smokers. It is estimated that by 2030, 1 in 1600 US adults will have a history of childhood KD.[12] In one analysis of individuals <40 years of age undergoing cardiac catheterization for suspected ischemia, 5% had CAA presumed or definitely secondary to KD.[13] This emphasizes the need for aggressive risk factor modification and longitudinal care.[11,14,15]

ACS IN THE ACUTE PHASE AND EARLY PEDIATRIC PERIOD

Approximately 81% of KD-related MIs occur within the first 2 years of onset, with the highest risk in the first 2 to 3 months.[2,6,16,17] Acute thrombotic coronary occlusions are the common cause. Thrombosis is promoted by a combination of prothrombotic factors: arterial wall inflammation, systemic platelet activation, and abnormal hemodynamics.[18,19] In giant aneurysms, decreased wall shear stress and blood stasis allow thrombi to form even during therapeutic antithrombotic therapy.

Clinical recognition in pediatric patients is difficult because classic symptoms are often absent.[20] Infants and young children may show nonspecific signs: unexplained irritability, vomiting, pallor, sweating, or shock. Older children may exhibit more typical symptoms, like chest pain or dyspnea, but up to 37% of MIs in children are silent, identified only through surveillance imaging.[20,21] Notably, many of these events occur while the child is at rest or asleep. Centers managing pediatric patients with KD with large CAAs must maintain a high index of suspicion and use a dedicated ischemia protocol.[3]

Management of acute MI is a medical emergency requiring urgent restoration of coronary flow (Figure 1).[2–4,22] In infants and young children, systemic thrombolysis using tissue-type plasminogen activator is often the first-line intervention.[2,3,17] Recent protocols combining tissue-type plasminogen activator with bivalirudin may offer thrombolysis with lower bleeding risk than heparin.[23] This is particularly relevant for infants whose vessel size may preclude direct catheter-based intervention or in centers where specialized pediatric interventional cardiology is not immediately available. Percutaneous coronary intervention (PCI) including intracoronary tissue-type plasminogen activator is an option for older children if specialized staff and appropriately sized equipment are available within 90 minutes. PCI in KD is technically demanding due to massive thrombus burden in giant aneurysms.[24,25] Coronary artery bypass grafting (CABG) is a definitive treatment for chronic stenosis, but rarely plays a role in acute MI management. Patients with refractory symptoms or significant myocardial dysfunction may require circulatory support or heart transplantation.

ANGIOGRAPHIC PHENOMENOLOGY OF ACS IN ADULTS AND PATHOLOGIC CHARACTERISTICS

Adults presenting with ACS after childhood KD exhibit a distinct angiographic and pathobiologic phenotype compared with patients with classic ASCAD (CENTRAL ILLUSTRATION and the Table). In the context of KD, remodeling of coronary arteries describes the process by which coronary arteries that previously had aneurysms appear to "normalize" in their internal luminal diameter through luminal myofibroblastic proliferation, layered mural thrombus organization, and intimal thickening, but the arterial wall architecture remains permanently abnormal.[16] CAAs in KD result from necrotizing arteritis

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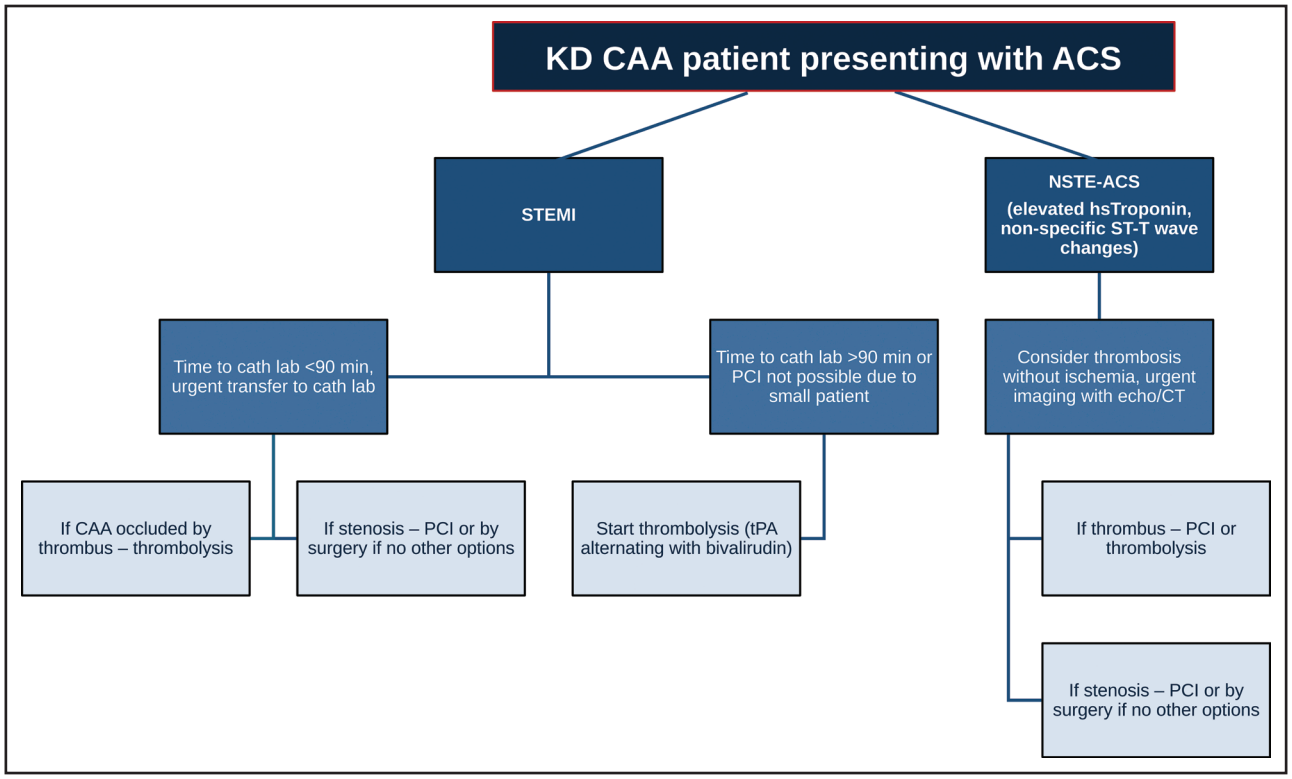

Figure 1. Management of patients with Kawasaki disease presenting with acute coronary syndrome.
CT indicates computed tomography; NSTE-ACS, non–ST-elevation acute coronary syndrome; PCI, percutaneous coronary interventions; STEMI, ST-segment–elevation myocardial infarction; and tPA, tissue-type plasminogen activator. Adapted from Jone et al.[2]

with destruction of the intima, media, and elastic laminae, leading to vessel wall weakening and outward dilation of the lumen, which can be fusiform or saccular.[16] The risk of ACS is highest in giant CAAs. A large circumferential proximal coronary artery calcification on fluoroscopy can provide the diagnosis of KD in adults presenting with ACS without a known history of KD.

Long-term coronary artery sequelae of KD (KDCAS) are predominantly characterized by CAA formation followed by maladaptive vascular remodeling, superimposed thrombosis, and progressive stenosis.[26,27] Culprit lesions in KDCAS underlying ACS most frequently involve the proximal epicardial coronary arteries, including the right coronary artery, proximal left anterior descending artery, and left main coronary artery. There is a strong association between CAA-specific morphologic characteristics—size, length, and number of persistent or regressed CAAs—and the localization of culprit lesions.[14,26–28] ACS in this population typically arises from either intraluminal thrombosis within aneurysmal segments or severe stenosis at the proximal or distal margins of aneurysms, where turbulent flow, endothelial dysfunction, and neointimal proliferation are most pronounced.[29,30] Unlike classical plaque rupture in ASCAD, lipid-rich atheroma is usually minimal or absent in children, although mild changes may be observed in younger adults.[29,31] Intracoronary optical coherence tomography has demonstrated that intimal and neointimal thickening is most pronounced in segments with persistent CAAs (Figure 2).[29]

Histopathologic findings and intravascular imaging consistently identify intimal hyperplasia as the dominant lesion, correlating with both historical maximum and current coronary dimensions. Fibrosis and cellular infiltration are observed across all categories of coronary involvement; more severe changes, such as media destruction, calcification, neovascularization, and white thrombi, are largely confined to segments with persistent or regressed CAAs.[32] High expression of genes associated with vascular remodeling suggests ongoing coronary arterial degeneration and features of vascular senescence long after the initial illness.[33] A high thrombus burden is a defining angiographic hallmark of KDCAS,[13] with the risk of thrombosis increasing substantially as Z scores rise above 20.[6] Angiography may demonstrate aneurysm segments with large, organized, or laminated thrombi, which may occur even after long periods of clinically quiescent disease. This process is likely driven by flow stasis within aneurysms and persistent endothelial dysfunction, often appearing disproportionate to the degree of fixed coronary stenosis.

Thrombotic occlusions within aneurysmal segments may undergo recanalization, often characterized by the development of multiple small, parallel channels within the original vessel lumen, known as a “lotus root” appearance.[34,35] These channels restore some degree of flow, but are vulnerable to reocclusion due

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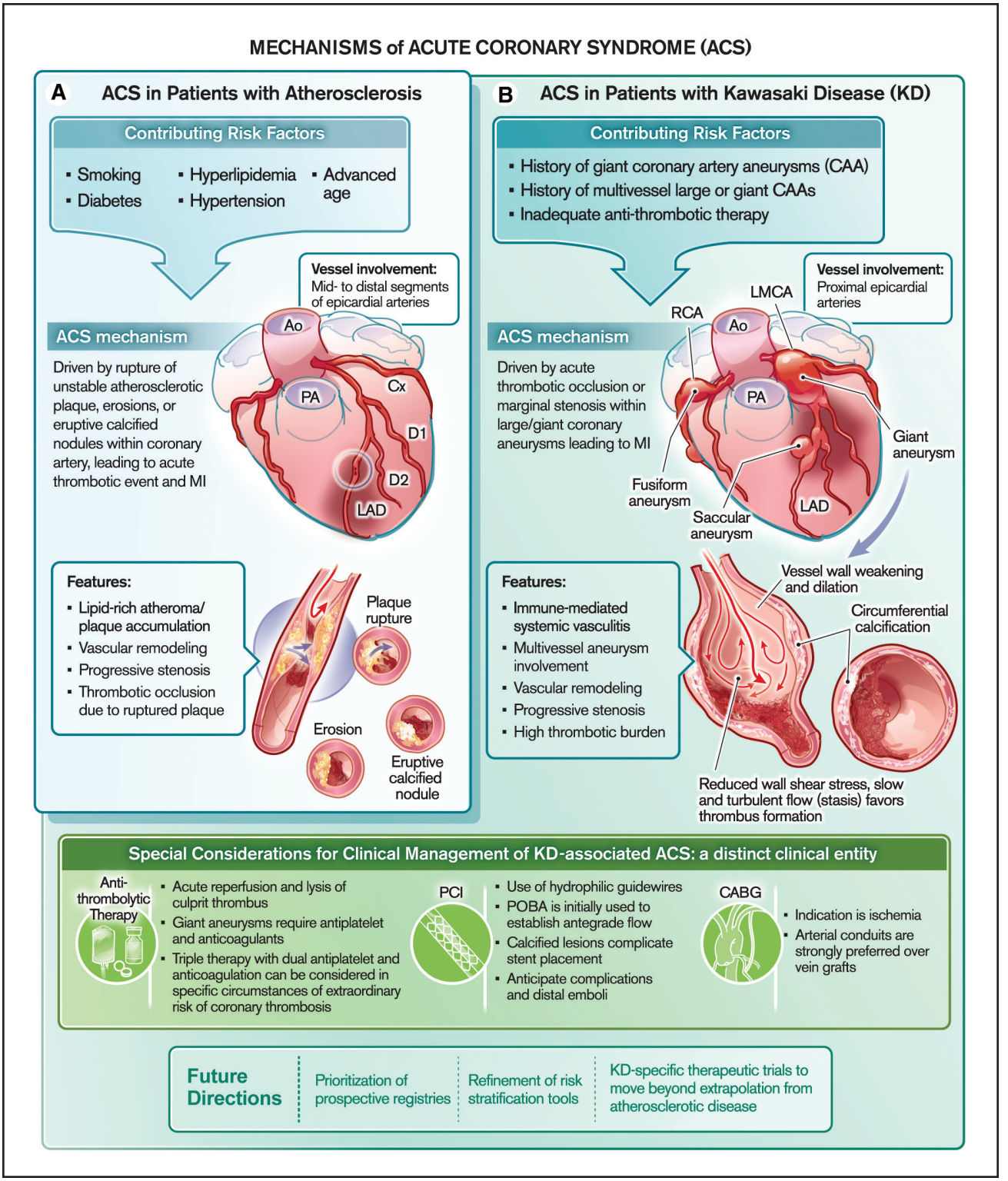

CENTRAL ILLUSTRATION. Mechanism of acute coronary syndrome in atherosclerosis versus Kawasaki disease.
This figure contrasts the distinct pathophysiologic pathways leading to myocardial infarction (MI) in patients with traditional coronary artery disease versus those with a history of Kawasaki disease (KD). Left, acute coronary syndrome (ACS) in atherosclerosis is driven by traditional cardiovascular risk factors, such as smoking, hyperlipidemia, hypertension, and advanced age. The primary mechanism involves the rupture or erosion of a lipid-rich atherosclerotic plaque, leading to acute thrombus formation, luminal occlusion, and subsequent myocardial damage (infarct). Right, ACS in KD stems from a pediatric history of KD and coronary artery aneurysms (CAAs). The mechanism is characterized by multivessel involvement and the formation of large or giant aneurysms across different territories. ACS typically occurs due to a large clot filling the aneurysm or calcified intramural disease rather than plaque rupture.

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Table. Comparative Features of Atherosclerotic Coronary Artery Disease and Kawasaki Disease–Associated Coronary Artery Sequelae

Domain	ASCAD	KDCAS
Disease paradigm	Chronic, lipid-driven inflammatory vascular disease	Immune-mediated childhood vasculitis with lifelong coronary sequelae
Primary coronary abnormality	Progressive atherosclerotic plaque formation	Coronary artery aneurysm formation with maladaptive remodeling
Dominant vascular process	Lipid accumulation, inflammation, and plaque progression	Intimal hyperplasia, fibrosis, and altered vessel architecture
Typical coronary morphology	Diffuse concentric or eccentric stenoses	Mixed aneurysmal and stenotic disease, often in the same vessel
Culprit lesion location in ACS	Variable; often mid-to-distal segments	Predominantly proximal epicardial arteries (RCA, proximal LAD, left main)
Mechanism of ACS	Plaque rupture or erosion with superimposed thrombosis	Thrombosis within aneurysms or stenosis at aneurysm margins
Thrombotic phenotype	Usually proportional to plaque burden	Frequently high thrombus burden, often disproportionate to fixed stenosis
Intravascular imaging signature	Thin cap fibroatheroma, plaque rupture or erosion	Marked intimal thickening, calcifications, organized thrombus
Aneurysm relevance	Rare, usually iatrogenic or after MI	Defining features: size, number, and persistence determine risk
Histopathologic hallmarks	Lipid-rich necrotic core, macrophages, calcification	Intimal hyperplasia, fibrosis, media destruction, limited lipid deposition
Calcification	Frequent and progressive	Mainly in persistent aneurysms or aneurysms that have regressed
Collateral circulation	Develops late in chronic ischemia	Often well-developed from childhood due to gradual obstruction
Disease evolution	Gradual plaque accumulation over decades	Dynamic remodeling: aneurysm persistence or regression, stenosis, thrombosis
Clinical course of ACS	Often isolated event related to focal plaque instability	Occurs on background of diffuse, chronic coronary abnormalities

ACS indicates acute coronary syndrome; ASCAD, atherosclerotic coronary artery disease; KDCAS, Kawasaki disease–associated coronary artery sequelae; LAD, left anterior descending; MI, myocardial infarction; and RCA, right coronary artery.

to progressive intimal thickening. Multivessel coronary involvement is prevalent; angiographic findings typically include multiple aneurysms across different coronary territories, frequently accompanied by mixed aneurysmal and stenotic diseases. Although ACS presentation is usually attributable to a single culprit vessel, extensive nonculprit coronary abnormalities are commonly present.[26,27]

Well-developed coronary collateral circulation is frequently observed in adolescents and young adults, reflecting a process of gradual coronary obstruction that typically begins in childhood and provides an extended window for adaptive remodeling.[36] Even in cases where angiography suggests a normal coronary artery, occlusion of small distal branches may only become apparent through collateral filling from contralateral vessels. This underscores the importance of careful angiographic assessment extending into the venous phase.[17] Despite robust collateral networks, patients with KD remain at risk for ACS; thrombotic occlusion or distal embolization can occur in segments beyond the reach of established collateral flow.

PATHOLOGIC CHARACTERISTICS OF CULPRIT LESIONS IN ADULT KD PATIENTS WITH ACS

Culprit lesions of ACS in adult patients with KD often present with high thrombus burden, mimicking the angiographic appearance of traditional atherosclerotic ACS.[15] However, the underlying pathophysiology of thrombosis is distinct. Whereas typical ACS is driven by plaque ruptures, erosions, or calcified nodules,[37,38] KD-related ACS arises from functional and structural abnormalities in the vessel wall, specifically from CAAs that formed during the acute phase of the disease.[15,32,39] These aneurysms result from inflammation-mediated damage, including necrosis of medial smooth muscle cells and disruption of the internal elastic lamina.[35,40] Although some CAAs regress, others persist, and can pose lifelong risks of MI. Large or giant aneurysms, defined as having a Z score of ≥10 or a diameter ≥8 mm, are associated with lifelong risk of cardiac events, including MI.[6,9,41] Intravascular imaging and histopathology often reveal layered mural thrombus, indicating that subclinical thrombosis occurs frequently in the absence of acute symptoms.[31,35,42]

Calcification and fibrosis are frequently observed in coronary arteries with CAAs in adult patients with KD using advanced imaging (eg, computed tomography [CT] calcium scoring,[43] coronary CT angiography [Figure 3],[44–46] intravascular ultrasound,[32] optical coherence tomography[31,47–49]) and pathologic investigations.[34,40] These changes likely represent a response to vessel inflammation, but the causal relationship between calcification or fibrosis and the occurrence of ACS in adult patients with KD remains unclear. In contrast, lipid plaques are rarely found in the coronary arteries of adult patients with KD.[31,47] This indicates that the pathogenesis of ACS in

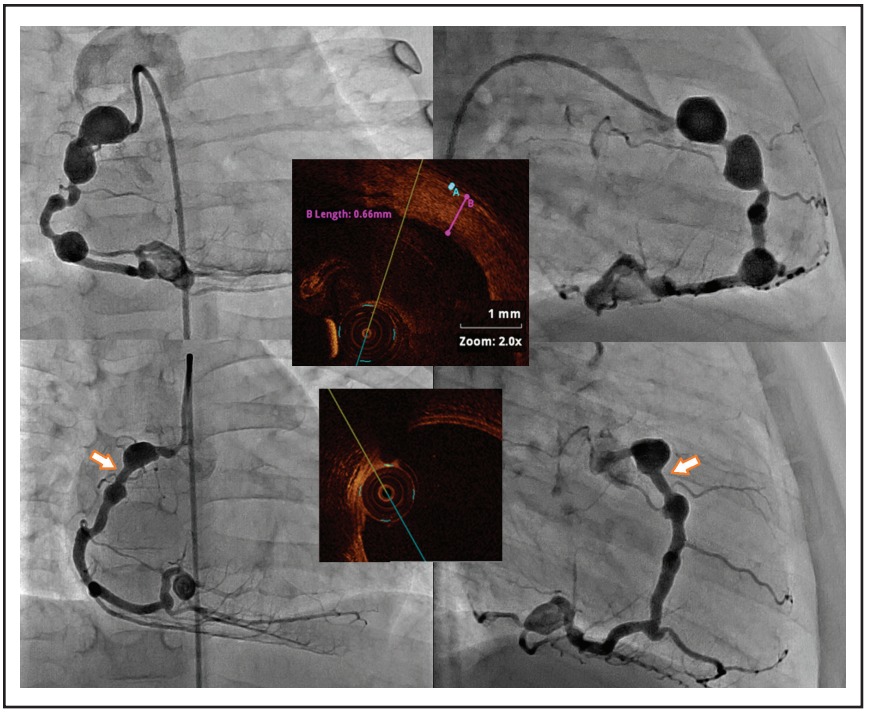

Figure 2. Optical coherence tomography on a regressed giant aneurysm site.
Optical coherence tomography (OCT) imaging (central insert) from a patient with multiple right coronary artery aneurysms (top, initial angiography) at the site of diameter regression (bottom, angiography comparison) of a giant aneurysm (arrows). The OCT reveals a heavily hyperplastic neoendothelial proliferation (B = measured 0.66 mm), histologically known to be generated by excess myofibroblastic proliferations. OCT imaging (bottom OCT insert) obtained from a normal never-aneurysmal coronary artery is provided for comparison (note the identical scale between the 2 OCT inserts and blue opposite poles lines around the circular catheter). In addition to the hyperplastic neoendothelial proliferation, an oval encapsulated calcified lesion is noted (adjacent to A).

adult patients with KD differs from that in typical patients with ASCAD, in which lipid-rich plaques are the main cause.

Evidence suggests that chronic inflammation may persist in the coronary arteries of patients with KD long after recovery from the acute illness; indeed, macrophage accumulation has been identified in CAA in both pathology and optical coherence tomography studies.[31,35,47] However, whether macrophages contribute to the development of ACS in adult patients with KD in the same manner as in atherosclerotic ACS is unclear. In addition, vasa vasorum in the intima and adventitia are common findings in pediatric and adult patients with KD with CAAs.[31,40,47] The vasa vasorum are generally thought to be linked to the progression of atherosclerosis,[50] but their role in patients with KD has yet to be clarified; they may promote vasculitis and CAA formation or, conversely, play a role in remodeling of CAAs.[51,52]

TECHNICAL CONSIDERATIONS FOR PCI IN ADULTS WITH PREVIOUS KD

PCI for ACS in adults with a history of KD (Figure 4, Figure S1, and Videos 1 and 2) is technically challenging due to the variable presence and severity of giant CAAs, fibrocalcification, and organized thrombus.[25,53] As with any PCI for ACS, the foremost goals include restoration of antegrade flow while minimizing embolic complications and long-term device-related risks. The 2025 guidelines for atherosclerotic lesions emphasize complete revascularization of clinically significant nonculprit lesions either during the index procedure or as a staged event, which may or may not be appropriate for KD.[4] Wiring strategies often require a departure from conventional ACS practices. In giant aneurysms, hydrophilic guidewires may help traverse thrombus-laden segments and distorted anatomy with ambiguous exit points, although they carry a risk of subintimal passage or perforation. Gentle wire

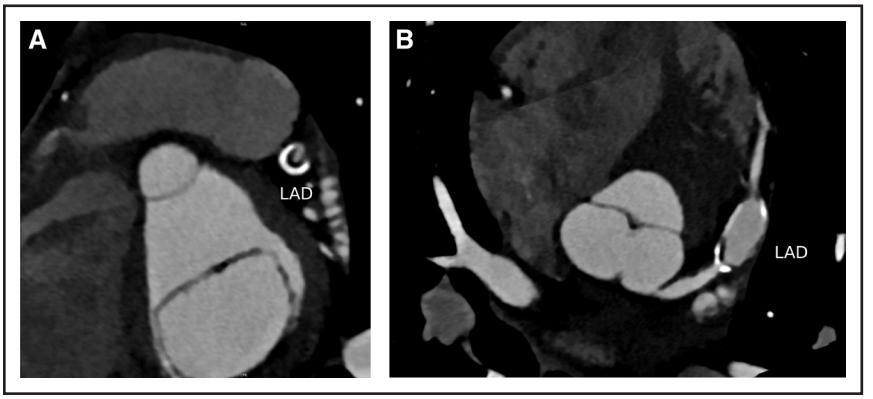

Figure 3. Coronary computed tomography angiography.
A, A cross-section of the proximal end of the aneurysm demonstrates near circumferential heavy calcification with a mural thrombus. **B**, A giant coronary artery aneurysm in the proximal left anterior descending (LAD) artery is calcified with a mural thrombus at the proximal end of the aneurysm.

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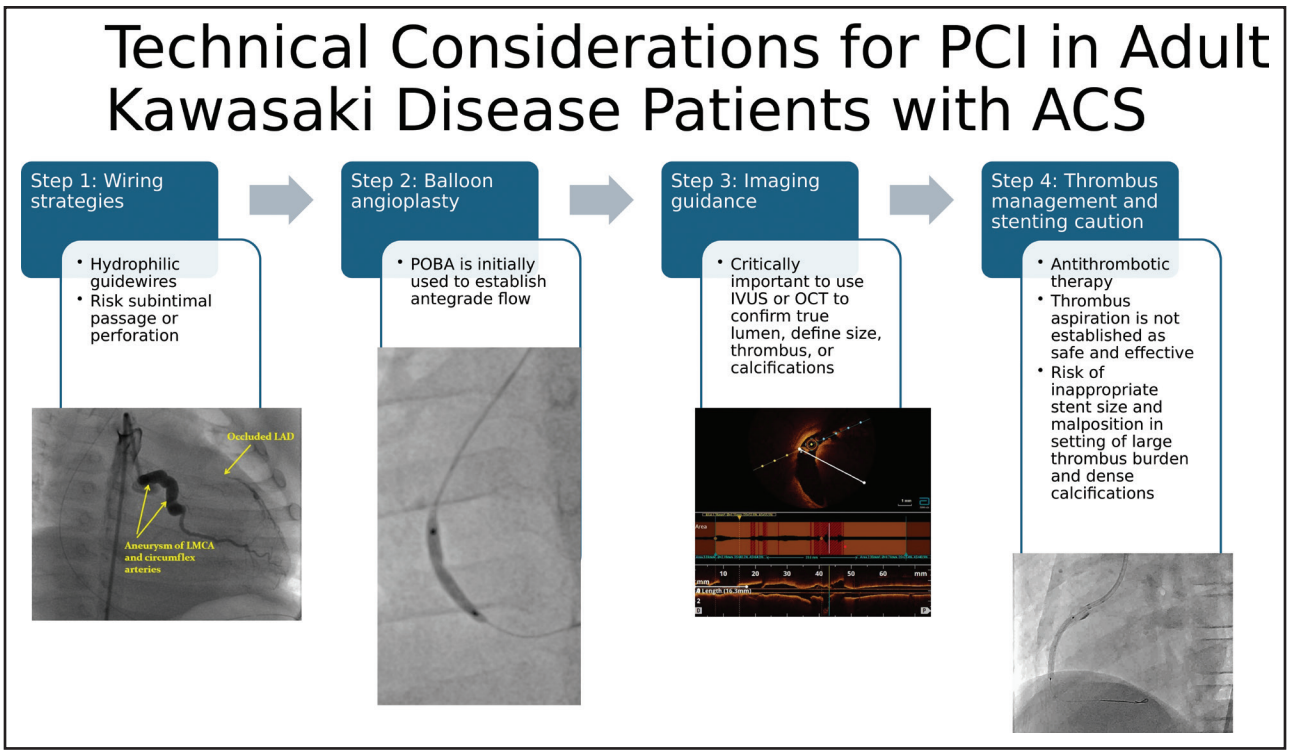

Figure 4. Key technical considerations for performing percutaneous coronary intervention in adult patients with coronary sequelae from childhood Kawasaki disease presenting with acute coronary syndrome.

The initial step involves selection of appropriate wiring strategies, including the use of hydrophilic guidewires. This is followed by balloon angioplasty. The third step emphasizes the use of intravascular imaging to guide the procedure. The fourth step outlines thrombus management focusing on antithrombotic therapy while noting that thrombus aspiration is not established as safe or effective. Furthermore, it highlights risks associated with stenting, including stent malposition and inappropriate stent sizing due to large thrombus burden and dense calcifications. Operators should anticipate potential complications and distal embolization. ACS indicates acute coronary syndrome; IVUS, intravascular ultrasound; LAD, left anterior descending; LMCA, left main coronary artery; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; and POBA, plain old balloon angioplasty.

manipulation with early confirmation of true-lumen position, ideally using intravascular imaging, is essential.

Balloon angioplasty is typically appropriate as the initial intervention once luminal wire position is secured. In some cases, it may be sufficient to restore antegrade flow, particularly when coupled with aggressive antithrombotic therapies. Historically, "plain old balloon angioplasty" has been favored over stents because metal stents may be undersized in lesions with large thrombus burden and may not be deployable in densely calcified lesions that cannot be dilated. Drug-eluting balloons represent an emerging technology of interest for which experience in KD lesions is lacking.[54] Options for PCI may be limited to balloon angioplasty in pediatric patients with smaller arteries and associated constraints on guide catheter caliber.

Given the complexity of these lesions, routine intravascular imaging is warranted to guide PCI and lesion preparation is recommended based on the 2025 ACS guidelines.[4] Intravascular imaging with intravascular ultrasound or optical coherence tomography is invaluable in KD-related ACS cases to define true vessel size, thrombus burden, presence and distribution of calcification, and postintervention results.[55]

Intracoronary stenting requires special caution in the KD context due to heightened risk of malposition, particularly when culprit lesions involve or abut aneurysmal segments. When target lesions encompass both aneurysmal and normal segments, stent sizing and postdilation strategies can be complicated, posing risks of edge dissection, geographic miss, distal embolization, impaired endothelialization, and late susceptibility to both restenosis and stent thrombosis. When angioplasty and medical therapy yield acceptable angiographic results, a decision to defer stent implantation may be preferred. Covered stents have been successfully deployed to exclude CAA, but in-stent restenosis has been an issue.[56,57] The long-term outcomes of this approach are not yet known.

Whereas routine use of manual aspiration thrombectomy is not recommended for PCI in ACS associated with ASCAD,[58] it may play a role in KD-related ACS associated with massive thrombus burden, particularly when initial balloon angioplasty fails to restore flow. However, the safety and success rate of this approach has not been established. In ASCAD, meta-analysis of thrombus aspiration in ST-segment–elevation MI does

not show a reduction in the risk of death, MI, or stent thrombosis, but is associated with a small increased risk of stroke.[59] Innovations in thrombectomy technologies include mechanical devices with continuous suction as well as stent-retriever devices that are subjects of active investigation with yet undetermined role in the patient with KD.[60,61] Laser vaporization of intracoronary thrombus uses a pulsed ultraviolet (xenon-chloride) laser that directly targets and vaporizes the thrombus. Excimer laser coronary angioplasty has been used for thrombosed culprit lesions in ST-segment–elevation MI, with mixed results.[62,63] The published experience with excimer laser coronary angioplasty in KD is limited to a single case report.[64] Because the culprit lesion in KD is frequently associated with dense calcification, advancement of the excimer laser coronary angioplasty catheter may be difficult or impossible. Intracoronary shockwave lithotripsy is an emerging tool that may play a role in circumferential aneurysmal calcification in KD.[65]

Distal embolization must be anticipated, and considered as a mechanism of no flow, slow flow, and no reflow beyond epicardial occlusion. Embolization and microvascular obstruction are often present at baseline and may be exacerbated by PCI. Distal embolization with slow flow due to microvascular obstruction, spasm, or edema can be approached pharmacologically with intracoronary adenosine, calcium channel blockers, nitroprusside, nicorandil, or epinephrine.[66] Glycoprotein IIb/IIIa inhibitors can also be considered. Nitroglycerin is less effective for true no reflow because it works mainly on epicardial arteries. Distal embolic protection devices (eg, filters) are conceptually attractive but empirically not validated in KD. Systemic or intracoronary thrombolysis remains an important adjunct or alternative when mechanical strategies are insufficient or anatomically constrained. Registry data from Japan indicate higher use of thrombolysis in KD-related ACS compared with ASCAD.[67]

A coronary chronic total occlusion (CTO) is defined as complete occlusion (100% stenosis) of a coronary artery with no antegrade flow (TIMI [Thrombolysis in Myocardial Infarction] flow grade 0) for a duration of ≥3 months based on angiographic evidence.[68,69] Because CTO develops gradually, the coronary capillary network often compensates by building collateral bridging vessels, and frequently evolves to maintain myocardial perfusion. CTOs are distinguished from acute total occlusions by their chronicity and pathologic composition. In contrast to atherosclerotic CTOs, KD-associated CTOs (Figure 5) are often characterized by extensive calcification, long-segment disease, organized thrombus with recanalized “lotus root” morphology, and well-developed collateral circulation. Therefore, PCI may be substantially more challenging than conventional CTO intervention, with lower procedural success rates and increased risk of complications. In asymptomatic patients with preserved ventricular function, normal stress imaging, and robust collateral circulation arising from coronary arteries free of significant KD-related pathology, conservative management is generally favored.

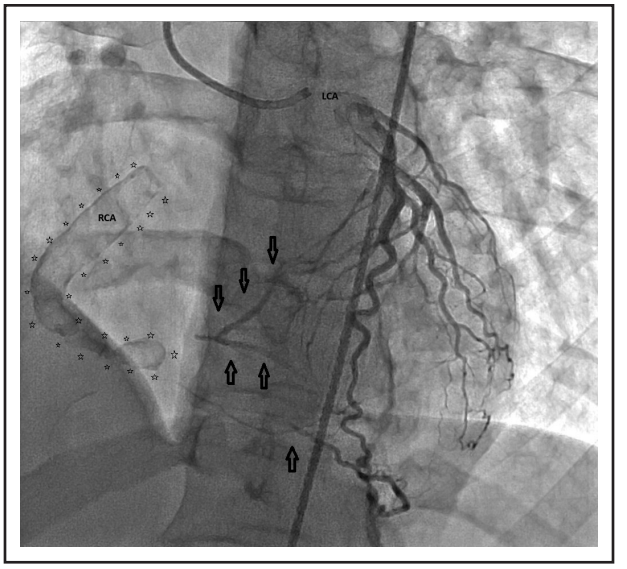

Figure 5. Left coronary artery angiography.
A selective left coronary artery angiography demonstrates patent left coronary circulation with rich collateral neovascularization (arrows) to an occluded right coronary artery with parietal macrocalcification (stars) along most of its length.

Revascularization should be guided by objective evidence of ischemia and myocardial viability rather than angiographic appearance alone and should not be undertaken solely because a CTO is present. The long-term stability of the collateral supply is an important consideration in clinical decision-making. Concern arises when the CTO territory is dependent on collateral vessels that may have clinically significant stenosis, progressive aneurysmal disease, or multiple CAAs that may predispose to future thrombosis. In such circumstances, revascularization may be considered even in the absence of demonstrable ischemia, particularly in younger patients with longer anticipated life expectancy. Conversely, when the collateral coronary artery is relatively preserved, collateral flow is robust, ventricular function is normal, and no objective evidence of ischemia is present, conservative management remains a reasonable strategy.

Overall, decisions regarding revascularization of KD-associated CTOs should incorporate not only symptoms, ischemia burden, ventricular function, and patient age, but also the integrity and anticipated durability of the collateral circulation. Given the complex anatomy, severe calcification, and unique lesion morphology frequently encountered in KD-associated CTOs, the procedural risks and likelihood of durable success must be carefully weighed against the potential benefits of intervention. If routine clinical surveillance reveals a new CTO in KD-related CAA, an ECG and cardiac enzymes should be obtained, the patient should be assessed for inducible

ischemia using functional stress testing, and their anticoagulation regimen should be reevaluated.

ANTITHROMBOTIC THERAPY AFTER ACS

Establishing an effective antithrombotic regimen after ACS in KD requires a nuanced approach that accounts for the patient's existing coronary anatomy and previous treatment history. For patients with large or giant CAAs, standard management typically involves systemic anticoagulation using warfarin, low-molecular-weight heparin (particularly in newly diagnosed pediatric cases), or direct oral anticoagulants. This anticoagulation is frequently combined with aspirin or dual antiplatelet therapy (DAPT). Although aspirin or DAPT is the primary indication for small or medium CAAs, clinicians may occasionally consider anticoagulation for aneurysms that have regressed to medium size but remain at high risk for luminal narrowing due to thrombosis.[2] In instances of aspirin hypersensitivity, clopidogrel or another P2Y12 inhibitor serves as an appropriate alternative.[2,16,17]

Antiplatelet Therapy

In adults with ASCAD and ACS, the standard of care involves DAPT—consisting of low-dose aspirin (75–162 mg for adults; 3–5 mg/kg for children) and a P2Y12 inhibitor (eg, clopidogrel, ticagrelor, prasugrel)—for 12 months.[4,70] This duration is intended to prevent recurrent thrombosis while balancing the inherent risk of hemorrhage. Depending on the individual's risk profile, DAPT courses may be shortened to 3 to 6 months or extended further. For long-term secondary prevention, although aspirin has historically been the gold standard, recent evidence suggests clopidogrel may offer a more favorable clinical profile.[71]

For patients with KD who receive drug-eluting stents during ACS treatment, DAPT is generally recommended for a full year.[4,70] In cases where the patient is already on anticoagulation, such as in patients with giant CAA, the primary challenge is balancing the risk of stent thrombosis against that of major bleeding. A regimen of "triple therapy"—combining anticoagulation with DAPT—may be used for up to 1 year in those with low risk of bleeding and high risk of recurrent coronary thrombosis. However, when the risk of serious bleeding is judged to be high, aspirin is dropped after 1 week, and an oral anticoagulant plus a P2Y12 inhibitor is continued for 12 months. Similarly, for those undergoing CABG, DAPT with aspirin together with a P2Y12 inhibitor should be reintroduced early postoperatively to ensure graft patency.[72] Aspirin alone may be sufficient after 6 to 12 months, but those with preexisting giant CAAs must continue their anticoagulation alongside antiplatelet therapy.[2,72] This same integrated protocol of DAPT with or without preexisting anticoagulation may be followed in patients with ACS who are treated with thrombolysis.[73]

Anticoagulant Therapy

Anticoagulation is indicated for patients with KD with giant CAAs after ACS, as failure to maintain this therapy has been linked to higher rates of recurrent events.[2,16,17,74] Because adults with atherosclerotic disease often face a high risk of bleeding events when on triple therapy (anticoagulation with DAPT), tools to stratify bleeding risk are used to optimize the net clinical benefit of antithrombotic treatment regimens.[72] Children and young adults appear to tolerate these combinations with a lower incidence of major bleeding.[75]

Warfarin has traditionally been the most frequently prescribed, but there has been a progressive shift toward direct oral anticoagulants in both adults and children because they require less invasive monitoring, are not affected by food with vitamin K, and carry a lower risk of vascular calcification.[2,76] Randomized trials of apixaban[77] and edoxaban[78] reported that bleeding and thromboembolism were uncommon. A series in predominantly adults with KD added to the literature on direct oral anticoagulant use in patients with giant aneurysms.[79] Furthermore, the availability of reversal agents, such as 4-factor prothrombin complex concentrate for apixaban and idarucizumab for dabigatran, provides a safety net for severe bleeding.

LIPID-LOWERING AND ANTI-INFLAMMATORY THERAPIES

Statins are used in KD for both their lipid-lowering capabilities and their pleiotropic, anti-inflammatory effects. They are a foundational therapy for treatment and secondary prevention in ACS in the general population,[80] but direct evidence in KD-associated ACS is lacking.[81,82] Despite this gap, the initiation of statin therapy is considered reasonable in patients with KD with CAA based on extrapolation of data from the general adult ACS population and the established benefits of reducing low-density lipoprotein cholesterol levels to prevent recurrent ischemic events.

In KD-associated ACS, coronary events most commonly result from thrombosis within CAAs rather than classic plaque rupture. Nevertheless, patients with previous KD may demonstrate persistent dyslipidemia and endothelial dysfunction, which may contribute to premature atherosclerosis.[83–85] Beyond cholesterol management, statins may provide disease-modifying benefits through anti-inflammatory mechanisms independent of lipid-lowering. Elevated inflammatory biomarkers, including high-sensitivity CRP (C-reactive protein), serum amyloid A, and calprotectin, have been reported in patients with persistent CAA.[39,86] Advanced imaging studies have demonstrated ongoing coronary arterial wall inflammation years after the acute illness.[87]

In a murine model of CAA, statin therapy suppressed inflammatory cell infiltration within the vascular wall.[88] Small clinical studies in patients with coronary aneurysms have demonstrated reductions in high-sensitivity CRP and improvements in endothelial function.[89,90] Imaging case reports have also suggested reductions in coronary inflammation with statin therapy, with recurrence after discontinuation.[91] However, statins were not routinely prescribed after ACS in a Japanese real-world cohort, highlighting variability in clinical practice.[11]

Colchicine represents an additional potential anti-inflammatory strategy. Low-dose colchicine has been shown to reduce major adverse cardiovascular events as secondary prevention in adults with coronary artery disease.[92] However, no studies have specifically evaluated colchicine in KD, and its safety and efficacy in this population remain uncertain.

CABG IN KD

CABG is an established revascularization strategy for both adults and children with KD.[93,94] For adolescents or adults, technical considerations include graft design, aneurysm intervention, approach, and postoperative medications. Although the optimal CABG graft design for KD is not fully established, longitudinal data provide insights. Tadokoro et al[95] analyzed a series of 92 patients with KD who underwent CABG between 1982 and 2017 at a mean age of 15 years. They concluded that the use of multiple arterial conduits, such as bilateral internal thoracic, radial, or gastroepiploic arteries, did not significantly alter overall survival, but resulted in fewer cardiac events compared with the use of a single arterial conduit. Given that multiple arterial conduits have been reported to yield better long-term outcomes in adults with atherosclerotic disease,[96] this concept may be applied to patients with KD. The series by Tadokoro et al[95] also reported absence of aneurysm rupture, suggesting that KD-associated CAAs, which typically develop a calcified wall over time, rarely rupture over the long term. Rather, thrombosis in the aneurysms is the major concern,[26] as 4 sudden deaths occurred within 6 years after the surgery. Therefore, a combined regimen of anticoagulation and antiplatelet therapy is essential after CABG in patients with residual giant CAAs. Furthermore, advanced surgical techniques, including minimally invasive, robotically assisted mini-left thoracotomy, have proven feasible and may shorten the hospital stay.[97]

A growing population of interest is adults who underwent CABG as children or adolescents presenting with ischemia in previously nonrevascularized territories. Most of these patients initially received a left internal mammary artery graft to the left anterior descending artery. Some patients had saphenous vein grafts to the right coronary artery or circumflex territories, which became stenosed or occluded. Managing these patients requires specialized expertise in complex, repeat surgical revascularization, particularly when navigating patent internal mammary artery grafts.

HEART TEAM AND SURVEILLANCE AFTER PCI AND CABG

Patients with KD with a history of ACS face increased risk for future cardiovascular events, including recurrent MI, heart failure, and death. Long-term data from Japan indicate a 30-year post-MI survival rate of 62.7% and a 25-year freedom from ventricular tachycardia rate of 28.5%. Higher risk of ventricular tachycardia is associated with lower left ventricular ejection fraction (LVEF) after MI and persistent myocardial perfusion abnormalities.[98]

After revascularization, patients require close monitoring and strict adherence to antithrombotic regimens. Revascularization addresses luminal stenosis, but CAAs may persist; therefore, long-term anticoagulation may still be required, based on aneurysm size and thrombotic risk.[2] β-Blockers and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers are standard in adult patients with previous MI and LVEF <40%,[99] and should be considered in pediatric patients after ACS.[2] Statins may be considered in children >2 years of age for their pleiotropic anti-inflammatory effects, although randomized controlled trials specifically supporting their efficacy in KD are lacking.[81,82] These findings emphasize the importance of lifelong cardiovascular surveillance and comprehensive secondary prevention in KDACS.

Although coronary artery abnormalities in KD arise through mechanisms distinct from atherosclerosis, patients with coronary abnormalities resulting from KD may also develop superimposed ASCAD. Therefore, long-term management should emphasize aggressive cardiovascular risk reduction, including blood pressure monitoring, lipid screening, and lifestyle counseling.[2] American Heart Association recommendations suggest that patients with KDCAS be treated at thresholds for those at high risk for premature atherosclerosis.[100]

Imaging surveillance in KD differs from the symptom-driven strategies in ASCAD.[99] Coronary CT angiography (Figure 6) can be used as surveillance imaging 1 year after PCI and CABG to assess CAAs to monitor for progression, recurrent thrombosis, future stenosis prognostication, mural changes, and additional calcifications.[45] Repeat coronary CT angiography every 3 to 5 years may be reasonable.[45] Functional surveillance with stress echocardiography, stress perfusion cardiac magnetic resonance imaging, or nuclear perfusion imaging are performed to identify inducible ischemia and may predict major adverse cardiovascular events in patients with KD.[101,102] Detailed surveillance strategies are outlined in the 2024 American Heart Association KD update and recent guidance on multimodality coronary

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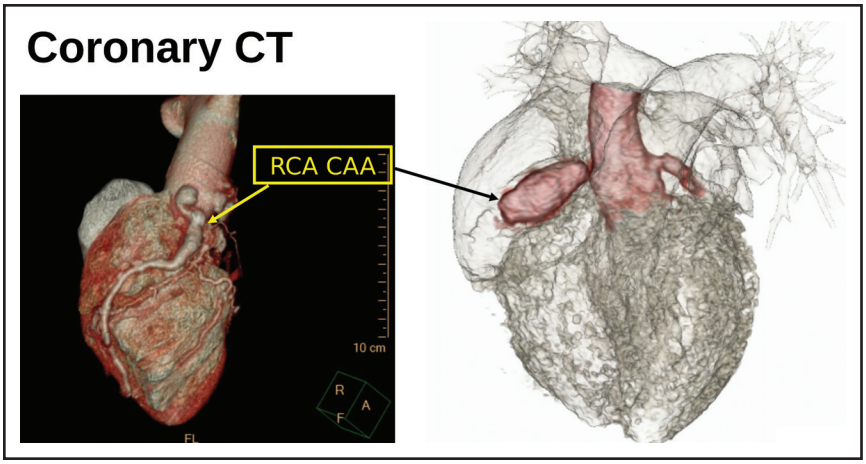

Figure 6. Three-dimensional reconstruction of coronary computed tomography angiography.
Scans from patients with Kawasaki disease and right coronary artery coronary artery aneurysm (RCA CAA): left, 41-year-old woman; right, 2-year-old girl.

ischemia testing.[2,103] Emerging imaging technologies may further enhance longitudinal assessment. Photon-counting detector CT offers improved spatial resolution and a superior ability to overcome calcification artifacts compared with conventional CT angiography.[104,105] This would be important in patients with KD with heavily calcified aneurysms to exclude coronary stenosis or to evaluate coronary stents after PCI, although no KD-specific photon-counting CT reports have been published to date. Ferumoxytol-enhanced coronary magnetic resonance angiography has been correlated with invasive catheter angiography for diagnosing coronary artery stenosis in KD. Coronary vessel wall imaging with 2- and 3-dimensional black blood turbo spin echo sequences are promising cardiac magnetic resonance imaging techniques that may allow direct visualization of vessel wall remodeling and monitoring of progressive intimal proliferation in patients with KD.[106]

Management of ACS in KD requires a lifelong, multidisciplinary heart team approach that integrates pediatric and adult cardiovascular expertise. Revascularization decisions should be individualized on the basis of lesion complexity, patient characteristics, and anticipated long-term outcomes, with coordinated postprocedural care including antithrombotic therapy, surveillance imaging, and cardiac rehabilitation. For adults presenting with non–ST-segment–elevation MI or unstable angina who are clinically stable, transfer to specialized centers with expertise in KD-related coronary interventions should be considered when feasible.[22] Elective revascularization is ideally performed at dedicated KD referral centers, where concentrated experience facilitates optimal patient care, systematic outcomes assessment, and evaluation of emerging technologies, such as intravascular lithotripsy, drug-coated balloons, and novel stent strategies.

PROGNOSIS AFTER PCI AND CABG

Reports on outcomes after PCI in patients with KD remain limited and are based on small patient cohorts. PCI is more frequently used than CABG to treat ASCAD, but its application in KD is restricted by the presence of complex giant aneurysms, extensive calcification, and a high propensity for recurrent stenoses necessitating reinterventions. These procedures are inherently more complex than standard interventions, often necessitating percutaneous transluminal coronary rotational atherectomy rather than plain old balloon angioplasty for treating severe, highly calcified coronary artery stenosis.[107] Data on long-term PCI outcomes are constrained by small cohort sizes, but successful interventions have demonstrated target vessel patency >10 years.[17,25,108–110] However, target-lesion revascularization rates remain significantly higher after PCI compared with CABG.[107,108,111]

In contrast, CABG has established an excellent track record of postoperative outcomes in patients with KD spanning >50 years.[108,112,113] A Japanese nationwide survey reported a 30-day operative mortality rate of <1%.[114] Factors associated with poor early outcomes include acute MI, heart failure, cardiogenic shock, need for additional cardiac mechanical support, history of an arrhythmia, low preoperative LVEF, New York Heart Association Class ≥III, and emergency surgery status. Outcomes for internal mammary artery grafts in children <12 years of age have improved with appropriate indications and plain old balloon angioplasty for anastomotic stenoses.[115,116] Grafts that remain patent with adequate flow 1 year after surgery typically remain patent for >20 years.[112] For adult patients with low LVEF, off-pump CABG has proven beneficial.[114,116]

The 10-year survival rate after CABG in patients with KD is high (≈94%), but the 10-year overall cardiac event–free survival rate, including repeat coronary revascularization after CABG, is only ≈66%.[114] Roughly 10% to 20% of patients will require a second coronary revascularization procedure within 10 years of their initial CABG. Furthermore, a recent review of severe KD-related cardiovascular events highlighted gaps in secondary prevention: one-quarter of patients with ACS and KD smoked, and fewer than one-third were discharged on statins after an ACS event.[11] Long-term prognosis after CABG is closely linked to LVEF and management of

cardiovascular risk factors. As this population ages, vigilant management of atherosclerosis risk factors is essential to lower the risk of progressive disease in nongrafted native coronary arteries and to maintain long-term graft patency.

KD PATIENT PASSPORT

Patient passports are being used in rare disease to help support communication of healthcare providers with patients and families.[117] Their use presents an especially important opportunity to improve care of patients with CAA secondary to KD. A health passport can help facilitate a structured healthcare transition for patients with KD as they move from parent-supervised pediatric care to independent adult management. The transition to adulthood is a period of high vulnerability; without a planned process, young adults often fall through the cracks of the healthcare system, leading to increased morbidity and mortality.[118] This risk is underscored by a survey in Japan, where >40% of patients with KD with coronary artery lesions were lost to follow-up.[119] A health passport bridges this gap by documenting clinical details that might otherwise be forgotten, such as the initial disease severity and maximal coronary internal diameter with Z scores. We hypothesize that inclusion in the passport of an image file containing the latest known coronary artery anatomy would enhance the success of timely, successful treatment in the event of an ACS.

The Taiwan Kawasaki Disease Children's Health Passport serves as a patient-held record that documents diagnosis, coronary status, and therapies, such as antiplatelet and anticoagulant treatment. It also provides a simplified framework for recommended surveillance and follow-up based on coronary involvement. Although not a substitute for individualized medical care, the passport can help families understand the importance of lifelong monitoring. By providing adult cardiologists with a concise summary of a patient's KD history and CAAs, the passport may facilitate timely recognition and treatment of ACS (KD Passport in the Supplemental Material). Ultimately, health passports can improve continuity of care and reduce lapses in follow-up across the lifespan.

CONCLUSION

ACS in patients with history of KD is a distinct clinical entity that differs fundamentally from ASCAD in pathophysiology, anatomy, clinical presentation, and management. Although the overall incidence of CAAs has declined in the era of timely intravenous immunoglobulin therapy, patients with large or giant aneurysms remain at lifelong risk for thrombosis, stenosis, MI, and sudden death. As this population ages, clinicians across pediatric and adult cardiovascular disciplines will increasingly encounter KD-associated ACS.

Optimal care requires early recognition of ACS symptoms in patients with a history of KD and understanding of the unique angiographic phenotype characterized by aneurysmal segments, high thrombus burden, calcification, and complex multivessel involvement. Revascularization strategies, whether thrombolysis, PCI, or CABG, must be individualized and guided by intravascular imaging whenever feasible. Antithrombotic regimens require careful balancing of thrombosis and bleeding risks, particularly in patients requiring combined anticoagulation and antiplatelet therapy. Secondary prevention should include aggressive cardiovascular risk factor modification.

Given the ongoing risk of major adverse cardiovascular events decades after the acute illness in patients with large or giant CAAs, lifelong surveillance, aggressive cardiovascular risk reduction, and structured transition from pediatric to adult care are essential to improve long-term survival. Future research should prioritize prospective registries, refinement of risk stratification tools, and KD-specific therapeutic trials to move beyond extrapolation from atherosclerotic disease. A disease-specific framework for the recognition, acute management, revascularization, antithrombotic therapy, and longitudinal surveillance of KD-associated ACS is key to improving outcomes in this growing population.

ARTICLE INFORMATION

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Supplemental Material

Kawasaki Disease Passport
Figure S1
Videos S1–S2

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