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Review

Thrombosis in Kawasaki Disease: Implications for Coronary Artery Aneurysm Risk, Diagnosis, and Anti-Thrombotic Management

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Abstract

Kawasaki disease (KD) is an acute systemic vasculitis that largely affects East Asian children, and these patients are at high risk for coronary artery aneurysms (CAAs), which predispose them to thrombotic events. Although the cause of KD remains unknown, the pathogenesis of thrombotic complications in KD appears to be complex and multifaceted, involving interactions between endothelial cell (EC) dysfunction, platelet activation, and immune cell interactions. In addition, KD can affect not only the coronary arteries but also the systemic vasculature, which can lead to thrombotic events beyond the coronary arteries. Early diagnosis and management of KD are essential to mitigate these risks, and new biomarkers, such as platelet function and leukocyte-platelet aggregates (LPAs), hemodynamics, are showing promise in identifying patients at high risk for thrombosis. In this review, we will explore the pathophysiologic mechanisms that contribute to thrombus formation in KD, review diagnostic strategies and emerging biomarkers, and discuss therapeutic options to prevent and manage KD-related thrombosis. A better understanding of these mechanisms may lead to improved approaches for early detection, risk assessment, and targeted therapy to decrease thrombotic complications in KD.

Keywords: Kawasaki Disease; Coronary Aneurysm; Thrombosis

Introduction

Kawasaki disease (KD) is an acute systemic vasculitis that predominantly occurs in children, with the highest incidence in East Asia. KD has a tendency toward the induction of inflammation in medium-sized blood vessels, especially in the coronary arteries. Without proper diagnosis and management, the risk of coronary artery aneurysms (CAAs) increases markedly, and progression to CAA can lead to serious cardiovascular complications such as thrombosis, stenosis, and myocardial infarction (MI), resulting in a poor prognosis.

For this reason, it is crucial to monitor patients and provide the right antithrombotic therapy carefully, but using these treatments without caution is not advised. To manage patients effectively, it is essential to categorize them based on their risk of developing CAA. Currently, the practice relies on the coronary artery diameter (D max) and Z-score to guide management. Notably, CAAs,

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which are caused by KD, contribute to posing a lasting cardiovascular risk, and studies show that a subset of young adults with acute coronary syndrome (ACS) have a history of KD-related CAAs, underscoring the disease's long-term effects. Despite these clinical risks, there is still a lack of clear understanding and consensus on the best way to prevent and manage thrombosis in KD patients. Many recommendations are based on retrospective analyses or on adult cardiovascular disease, leaving critical gaps in evidence for pediatric patients. This uncertainty highlights the urgent need to understand the mechanisms behind thrombus formation in KD and to develop novel diagnostic tools and therapeutic strategies tailored to its unique pathophysiology.

The purpose of this review is to provide a comprehensive overview of the underlying causes, clinical features, diagnostic methods, and treatment options for thrombosis in KD. Lastly, this review focuses on the latest developments in biomarker research, new diagnostic approaches, and anti-thrombotic management to enhance risk assessment and long-term care.

1. Pathobiology of thrombo-inflammation in Kawasaki disease

Thrombosis in KD occurs through a complex interplay of pathological changes, including vasculitis, endothelial cell (EC) dysfunction, hemodynamic changes, and platelet activation. These factors all contribute to a prothrombotic environment in patients with KD [1–3]. Specifically, the progression of acute inflammatory responses activates and damages ECs, compromising the anticoagulant and antiplatelet properties of the endothelium, thereby increasing the risk of thrombus formation [4,5]. In addition, the infiltration of immune cells and the excessive production of pro-inflammatory cytokines further amplify vascular inflammation. Also, interactions between platelets and immune cells play a crucial role in promoting thrombus formation [6].

1) Vasculitis and platelet activation

During the acute phase of KD, platelets become overactive, causing them to express P-selectin (CD62P) and release inflammatory mediators such as platelet factor 4 (PF4), β-thromboglobulin (β-TG), and CD42b/GPIB alpha [3]. Importantly, when platelet P-selectin interacts with PSGL-1 on leukocytes, it activates MAC-1, promoting the formation of leukocyte-platelet aggregates (LPA) [7]. As activated platelets bind to leukocytes to form these aggregates, they create a positive feedback loop [8]. These aggregates adhere to ECs, inducing vascular injury and disrupting hemodynamic stability. This interaction also triggers the expression of Tissue Factor (TF) on leukocytes, which, by binding with Factor VIIa, initiates the coagulation cascade and facilitates thrombus formation [9]. Furthermore, pro-inflammatory cytokines such as interleukin (IL)-1β, IL-6, and tumor necrosis factor (TNFα) are released during this process, fostering an inflammatory environment that promotes thrombosis progression [10]. The cytokine release establishes a pro-thrombotic and inflammatory state that exacerbates vascular complications in KD, particularly by affecting the coronary artery wall and leading to necrotizing arteritis during the acute phase [11]. This process involves the infiltration of neutrophils and macrophages, resulting in EC injury. Consequently, this

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inflammatory environment in KD extends beyond a simple inflammatory response and serves as a pathological mechanism contributing to thrombotic risk.

2) Endothelial cell dysfunction

EC dysfunction is a pivotal component of the pathophysiology of KD, significantly contributing to the development of CAA and thrombosis. During the acute phase of KD, pro-inflammatory cytokines, such as IL-1, IL-6, and TNF-α, activate ECs through the NF-κB signaling pathway [12]. As a result, activated ECs upregulate adhesion molecules, such as VCAM-1 and ICAM-1, facilitating the adhesion and transmigration of immune cells to the vascular wall. Furthermore, the activation of caspase-3 and caspase-7 induces EC apoptosis in ECs, while an additional form of inflammatory cell death, pyroptosis, is initiated by NLRP3 inflammasome activation, which is mediated by the HMGB1/RAGE/cathepsin B signaling pathway [13].

Endothelial-to-mesenchymal transition (EndoMT) has been observed in KD, characterized by a reduction in EC markers (VE-cadherin, ZO-1, Claudin-1) and an increase in mesenchymal cell markers (Vimentin, α-SMA, β-catenin). This transition leads to structural and functional abnormalities in blood vessels [13]. Notably, EndoMT has been found to promote vascular wall thickening and CAA formation in KD patients, as well as to stimulate the production of pro-inflammatory cytokines, such as IL-17, IL-6, granulocyte-macrophage colony-stimulating factor (GM-CSF), and vascular endothelial growth factor (VEGF), by transdifferentiated mesenchymal cells. These cells, in turn, have been shown to recruit various immune cells, including neutrophils, CD8+ T cells, and M1 macrophages [13]. EndoMT plays a crucial role in EC injury and CAA formation during the acute phase of KD, potentially increasing the risk of long-term cardiovascular complications [13]. These findings highlight the importance of EndoMT in the development of KD, suggesting its potential as a biomarker for KD diagnosis and a target for therapeutic intervention.

2. Coronary complications: coronary artery aneurysm (CAA) and thrombosis

1) Coronary thrombosis

KD is frequently complicated by CAA, occurring in about 25% of untreated patients but only 4% with prompt intravenous immunoglobulin (IVIG) [11]. These aneurysms disturb flow and shear and, together with endothelial injury, promote platelet activation and thrombosis, leading to occlusion, MI, distal embolization, or sudden events [9,14–16]. Risk and management are guided by Z-score (small 2.5–5, medium 5–10, giant ≥ 10); giant CAAs carry ~50% risk of future coronary events, for which long-term low-dose aspirin is recommended in small/medium lesions and additional anticoagulation [warfarin or direct oral anticoagulants (DOACs)] is considered for large or multiple aneurysms, alongside sustained cardiovascular surveillance [15,17].

In a cohort of 2,680 patients with KD, 500 developed CAA, and 90 of these were giant. Giant CAA was associated with poor prognosis, with higher rates of thrombotic occlusion,

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progressive stenosis requiring revascularization, and ACS during 30 years of follow-up. More than 20% experienced major adverse cardiac events, and about 25% regressed; therefore, ongoing cardiovascular monitoring is required [18–20]. Consistent with these findings, over half of patients with giant CAAs experience thrombotic events, highlighting the need for lifelong cardiovascular surveillance [11,21].

Distribution analyses consistently show a predilection for the right coronary artery (RCA) and left anterior descending artery (LAD): in one cohort of 94 KD-CAA cases, 23, 79, and 53 cases were identified in the LMCA, RCA, and LAD respectively, with coronary thrombosis in 54 patients (57.4%) and particularly high cumulative involvement of the LAD; in a separate coronary CT angiography (CCTA) series (23 patients), 60 CAAs were detected (37 proximal and 23 distal), again with proximal RCA and LAD most frequently affected [21,22]. A detailed analysis of the proximal CAAs revealed 15 cases in the RCA, 14 in the LAD, and 4 in the left circumflex artery (LCX).

In summary, RCA and LAD are the most commonly affected sites of CAA in KD patients, whereas LMCA and LCX show a relatively lower prevalence. This difference could be attributed to the higher wall shear stress and uneven hemodynamics observed in the LAD and RCA, which may be major contributors to the development of CAA and thrombosis. These features underscore critical pathophysiological aspects of KD and highlight the need for further research to elucidate these mechanisms and improve clinical management strategies [23,24].

2) Thrombosis in other vessels

Although CAAs are the most recognized vascular complication of KD, KD-induced vasculitis is not limited to the coronary arteries. It can also affect other systemic arteries, including the aorta, carotid, pulmonary, renal, and axillary arteries [25]. This systemic vascular involvement suggests that thrombotic events in KD should not be confined solely to the coronary arteries, but must be evaluated from a broader, systemic perspective. Interestingly, infants and IVIG-resistant patients are more likely to exhibit a higher incidence of non-coronary vascular involvement [26].

Peripheral artery thrombosis (PAT), in-situ thrombotic occlusion of non-coronary systemic arteries, predominantly in the limbs and trunk, is an infrequent complication of KD that has been documented in the literature. For example, a case study reported an infant with KD who developed a systemic arterial aneurysm complicated by arterial thrombosis, successfully managed through interventional procedures [26]. Another report described peripheral gangrene resulting from PAT in a KD patient, emphasizing the potential for KD to cause significant thrombotic complications outside the coronary arteries [27].

Reports of aortic thrombosis in KD patients have raised concerns regarding severe complications, such as aortic dissection and rupture, both of which are life-threatening [28]. In addition to aortic involvement, KD has also been linked to cerebrovascular complications, albeit less frequently. Cerebral infarction, a rare occurrence, has been documented as a complication during the acute or subacute phase of the disease [29]. Furthermore, mounting

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evidence points to a potential association between KD and cerebrovascular lesions, suggesting an elevated risk of ischemic stroke in children and young adults with a history of KD [30]. These findings underscore the necessity for comprehensive vascular assessment in KD patients, particularly those with severe or IVIG-resistant disease. Given the potential for both coronary and systemic thrombotic complications, early diagnosis and appropriate management remain essential, and evaluation of systemic arteries should be considered in patients who show resistance to standard therapy.

3. Diagnosis of coronary artery aneurysm (CAA) in Kawasaki disease

The primary diagnostic modalities for detecting CAAs, a major complication of KD, include echocardiography, CCTA, cardiac MRI (CMR), and invasive coronary angiography (ICA) [11,31,32]. Echocardiography is regarded as the optimal imaging modality for the diagnosis and monitoring of CAAs due to its non-invasive nature, its lack of ionizing radiation, and its capacity for serial evaluation. Consequently, it is considered the primary imaging technique for identifying CAAs in KD patients [33]. It is recommended that echocardiography be performed within the first week of KD onset for CAA diagnosis, followed by serial assessments [17]. However, its limitations include the difficulty of evaluating distal coronary arteries and a high degree of operator dependency.

CCTA has been shown to possess a higher degree of sensitivity than echocardiography in the diagnosis of CAAs. According to the research by van Stijn et al. [31], CCTA successfully identified 34 additional CAAs (56%) that were not detected by echocardiography. Plus, CCTA provides full visualization of the coronary artery tree, enabling the identification of left circumflex CAAs that are challenging to assess with echocardiography [34].
The comprehensive nature of CCTA allows for detailed evaluation of CAA size, wall characteristics, structural integrity, and lumen features, making it advantageous for Z-score measurement [32]. However, its limitations include radiation exposure and the risk of adverse reactions to contrast agents. Given these characteristics, CCTA is particularly useful for long-term follow-up of KD patients and is recommended as an adjunct imaging modality, especially for patients in whom echocardiography has already identified CAA lesions.

CMR is a non-invasive and radiation-free imaging modality that utilizes detailed visualization of the proximal and certain distal coronary arteries. It enables comprehensive assessment of the coronary lumen and vessel wall, myocardial inflammation, microvascular disease, MI, left ventricular dysfunction, and other cardiac pathologies, including CAA [35]. Myocardial stress CMR is a promising technique; however, further research is required to establish its role in routine evaluation and risk stratification of KD patients with coronary involvement.

Lastly, ICA presents high-resolution visualization of coronary flow and luminal integrity, thereby enabling confirmation of thrombus formation and differentiation from myointimal proliferation. It serves as a critical diagnostic tool for evaluating complex or refractory K KD cases, particularly when noninvasive imaging yields inconclusive results or fails to provide adequate visualization, and when therapeutic interventions such as revascularization are being

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considered.

Across these modalities, each imaging technique offers distinct strengths and limitations in the evaluation of CAAs in KD. Echocardiography remains central for initial assessment and routine monitoring, while CCTA and CMR provide more detailed anatomical and functional information when needed. ICA retains a crucial role in complex or uncertain cases. Together, these complementary approaches highlight the importance of selecting an appropriate imaging strategy tailored to disease severity and clinical context.

1) Biomarkers of Kawasaki disease

Clinically established biomarkers such as NT-proBNP/BNP and ferritin have been identified, which may help predict complications and assess risk in KD patients. NT-proBNP/BNP is a representative indicator reflecting myocardial stress, confirmed to be significantly elevated in acute KD patients [36,37]. This indirectly reflects myocardial inflammation and transient ventricular dysfunction occurring in KD. Ferritin is a protein that increases during acute inflammation, particularly elevated in severe KD patients [38]. Elevated serum ferritin levels are associated with an increased risk of cardiovascular complications, particularly CAA, in KD patients, making ferritin a promising biomarker for assessing disease activity and predicting prognosis [39,40].

Additionally, new potential biomarkers are actively reported in academic circles. The IL-17 family reflects activation of the Th17 axis, which plays a critical role in the pathophysiology of KD; elevated levels of IL-17A are observed in the acute phase, and these levels are particularly increased in patients with coronary artery lesions, underscoring its potential as a biomarker for vascular complication risk [41,42]. Finally, hepcidin is a hormone that regulates iron metabolism and tends to increase during acute inflammation, as observed at high levels in the serum of KD patients [43]. Hepcidin is a novel candidate indicator that could explain KD pathophysiology from an immunometabolic perspective, holding significant potential for future use in prognosis prediction and research on pathological mechanisms [44].

In conclusion, the clinical indicators and existing biomarkers identified to date can be effectively utilized for KD diagnosis and risk assessment. They may further contribute to predicting thrombotic complication associated with KD. However, new candidate biomarkers such as the IL-17 family and hepcidin are still based on limited research findings and require validation through large patient cohorts and long-term follow-up studies. Therefore, a multidisciplinary approach integrating clinical diagnosis and biomarker research will lay a crucial foundation for understanding KD pathophysiology and establishing new diagnostic and prognostic prediction systems.

4. Anti-thrombotic management

The standard treatment for acute KD is aspirin and IVIG. Aspirin use is the only class I recommendation among the pharmacological treatment of KD, but there is no international standard regimen [11]. According to the 2024 American Heart Association (AHA) guidelines, aspirin should be given at a moderate dose of 30–50 mg/kg or a high dose of 80–100 mg/

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kg daily. Whereas the 2020 Japan Circulation Society (JCS) guidelines recommend oral administration of a moderate dose of aspirin three times daily [45,46]. Both guidelines suggest patients should be given low-dose aspirin (3–5 mg/kg) daily after fever subsides, but AHA suggests a treatment duration of 4–6 weeks, while JCS recommends 2–3 months [45,46]. If the patient is unresponsive or allergic to aspirin, clopidogrel can be used as an alternative [11].

IVIG is infused at a dose of 2 g/kg over 8–12 hours to reduce inflammation and lower the risk of CAA [45]. If the patient experiences a persistent or recurring fever for 36 hours or more after the infusion of the initial IVIG treatment, they are considered resistant to IVIG [45]. A Japanese trial reported that corticosteroid therapy reduced the IVIG resistance rate and lowered the risk of CAA; however, there is no evidence that this is effective in non-Japanese populations [47]. Other drugs such as infliximab, a monoclonal antibody that targets tumor necrosis factor-alpha; anakinra, a recombinant IL-1 receptor antagonist; and cyclosporine, a calcineurin inhibitor that blocks T-cell activation, are being studied for use in IVIG-resistant patients, but current evidence is limited [48–50]. More randomized controlled trials are needed to standardize these and use them in clinical practice.

Antiplatelet medications are typically used in patients with medium and giant CAA, and anticoagulants are added in patients with giant CAA or a prior history of MI [11]. The most common combination is low-dose aspirin and warfarin, but warfarin poses a high bleeding risk and requires individualized dose adjustments. Currently, DOACs are under trial to validate their safety and effectiveness in KD patients [51]. Because thrombus formation within CAA drives many acute coronary events, long-term care should explicitly target clot prevention and early detection, for example, using regular imaging (echocardiography with CCTA/CMR as needed) to look for mural thrombus or evolving stenosis, and adding functional testing when ischemia is suspected. At each visit, antithrombotic treatment and bleeding risk should be reassessed. If thrombosis is suspected or confirmed, prompt anticoagulation and reperfusion strategies—thrombolysis, percutaneous coronary intervention, or coronary artery bypass grafting—should be considered based on clinical status and institutional expertise [46]. This integrated plan, which includes ongoing imaging, timely adjustments to antithrombotic treatment, and rapid intervention when clots occur, aims to prevent thrombosis and improve long-term outcomes in KD.

Conclusion

Thrombosis in KD reflects thrombo-inflammation triggered by endothelial injury, platelet–immune activation, and abnormal hemodynamics within CAAs. Endothelial dysfunction—including EndoMT with downstream IL-17–family signaling—weakens antithrombotic surface properties, while aneurysm-related flow disturbance creates a pro-coagulant environment that promotes platelet adhesion and activation. This mechanistic framework explains the clinical pattern: the coronary circulation is at high risk for thrombosis, and once CAA forms, disturbed shear stress and damaged endothelium promote local thrombus formation, distal embolization, and MI. With timely IVIG, CAA formation decreases from about 25% to 4%. However, once CAA is present, risk scales with size. Z-score categories

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(2.5–5 small, 5–10 medium, ≥ 10 giant) separate risk levels, and giant CAAs approach a 50% rate of coronary events—supporting stronger antithrombotic treatments and tight long-term monitoring.

Based on this KD pathology, it is imperative to advance research that clarifies KD's thrombo-inflammatory pathways and translates them into precise diagnostic criteria and effective, risk-aligned therapy. A main goal is the development of specific biomarkers that (i) enable early and accurate diagnosis—particularly when KD mimics other febrile illnesses—and (ii) identify thrombotic risk early enough to trigger prophylactic antithrombotic therapy and tighter surveillance. Objective, reproducible diagnostic and risk-stratification biomarkers would meet a major unmet need by enabling earlier intervention and reducing severe complications—CAA progression, in-situ coronary thrombosis, and MI. Such progress would not only lower KD-related mortality and lifelong thrombotic sequelae through prevention but also lessen the healthcare burden borne by patients, families, and systems.

References

1. Gedalia A. Kawasaki disease: 40 years after the original report. Curr Rheumatol Rep. 2007;9:336-41.
2. Seo J. Advancing risk stratification of coronary artery aneurysms caused by Kawasaki disease using hemodynamics analysis and computational fluid dynamics. Kawasaki Dis. 2023;1:e6.
3. Arora K, Guleria S, Jindal AK, Rawat A, Singh S. Platelets in Kawasaki disease: is this only a numbers game or something beyond? Genes Dis. 2020;7:62-6.
4. Bochenek ML, Schäfer K. Role of endothelial cells in acute and chronic thrombosis. Hamostaseologie. 2019;39:128-39.
5. Chanchal S, Mishra A, Singh MK, Ashraf MZ. Understanding inflammatory responses in the manifestation of prothrombotic phenotypes. Front Cell Dev Biol. 2020;8:73.
6. Rayes J, Bourne JH, Brill A, Watson SP. The dual role of platelet-innate immune cell interactions in thrombo-inflammation. Res Pract Thromb Haemost. 2020;4:23-35.
7. Cerletti C, Tamburrelli C, Izzi B, de Gianfagna F, Gaetano G. Platelet-leukocyte interactions in thrombosis. Thromb Res. 2012;129:263-6.
8. Zhang Y, Jia C, Guo M, Chen Q, Wen Y, Wang T, et al. Platelet–monocyte aggregate instigates inflammation and vasculopathy in Kawasaki disease. Adv Sci. 2025;12:e2406282.
9. Wagner DD, Burger PC. Platelets in inflammation and thrombosis. Arterioscler Thromb Vasc Biol. 2003;23:2131-7.
10. Yan SLS, Russell J, Granger DN. Platelet activation and platelet-leukocyte aggregation elicited in experimental colitis are mediated by interleukin-6. Inflamm Bowel Dis. 2014;20:353-62.
11. 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:e927-99.
12. Leung DYM. The potential role of cytokine-mediated vascular endothelial activation in the pathogenesis of Kawasaki disease. Paediatr Int. 1991;33:739-44.

https://doi.org/10.59492/kd.2025.3.2.e12

13. Jia C, Zhang J, Chen H, Zhuge Y, Chen H, Qian F, et al. Endothelial cell pyroptosis plays an important role in Kawasaki disease via HMGB1/RAGE/cathespin B signaling pathway and NLRP3 inflammasome activation. Cell Death Dis. 2019;10:778.
14. Kuramochi Y, Ohkubo T, Takechi N, Fukumi D, Uchikoba Y, Ogawa S. Hemodynamic factors of thrombus formation in coronary aneurysms associated with Kawasaki disease. Pediatr Int. 2000;42:470-5.
15. Miura M, Kobayashi T, Kaneko T, Ayusawa M, Fukazawa R, Fukushima N, et al. Association of severity of coronary artery aneurysms in patients With Kawasaki disease and risk of later coronary events. JAMA Pediatr. 2018;172:e180030.
16. Sideris G, Sideris G, Magkoutis N, Voicu S, Kang C, Bonneau M, et al. A novel experimental thrombotic myocardial infarction and primary angioplasty model in swine. EuroIntervention. 2019;14:e1843-51.
17. Kim SH. Diagnosis of coronary artery abnormalities in Kawasaki disease: recent guidelines and z score systems. Clin Exp Pediatr. 2022;65:430-8.
18. 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;5:1-18.
19. Suda K, Iemura M, Nishiono H, Teramachi Y, Koteda Y, Kishimoto S, et al. Long-term prognosis of patients with Kawasaki disease complicated by giant coronary aneurysms: a single-institution experience. Circulation. 2011;123:1836-42.
20. Elias MD, Brothers JA, Hogarty AN, Martino J, O'Byrne ML, Patel C, et al. Outcomes associated with giant coronary artery aneurysms after Kawasaki disease: a single-center United States experience. J Pediatr. 2024;274:114145.
21. Peng Y, Yi Q. Incidence and timing of coronary thrombosis in Kawasaki disease patients with giant coronary artery aneurysm. Thromb Res. 2023;221:30-4.
22. Singhal M, Pilania RK, Jindal AK, Gupta A, Sharma A, Guleria S, et al. Distal coronary artery abnormalities in Kawasaki disease: experience on CT coronary angiography in 176 children. Rheumatology. 2023;62:815-23.
23. Spaan JAE, Piek JJ, Hoffman JIE, Siebes M. Physiological basis of clinically used coronary hemodynamic indices. Circulation. 2006;113:446-55.
24. Soulis JV, Farmakis TM, Giannoglou GD, Louridas GE. Wall shear stress in normal left coronary artery tree. J Biomech. 2006;39:742-9.
25. Takahashi K, Oharaseki T, Yokouchi Y, Hiruta N, Naoe S. Kawasaki disease as a systemic vasculitis in childhood. Ann Vasc Dis. 2010;3:173-81.
26. Ozdemir E, Peterson RE. Systemic arterial aneurysm complicated by thrombosis in an infant with resistant Kawasaki disease. Ann Pediatr Cardiol. 2019;12:147-9.
27. Zhang N, Yu L, Xiong Z, Hua Y, Duan H, Qiao L, et al. Kawasaki disease complicated by peripheral artery thrombosis: a case report and literature review. Pediatr Rheumatol. 2022;20:77.
28. Hakim K, Rafik B, Lilia C, Fatma O. Giant aortic arch aneurysm complicating Kawasaki's disease. Ann Pediatr Cardiol. 2014;7:201-3.

https://doi.org/10.59492/kd.2025.3.2.e12

29. Wang L, Duan H, Zhou K, Hua Y, Liu X, Wang C. Kawasaki disease complicated by late-onset fatal cerebral infarction: a case report and literature review. Front Pediatr. 2021;9:598867.
30. Yeom JS, Cho JY, Woo HO. Understanding the importance of cerebrovascular involvement in Kawasaki disease. Korean J Pediatr. 2019;62:334-9.
31. van Stijn D, Planken N, Kuipers I, Kuijpers T. CT angiography or cardiac MRI for detection of coronary artery aneurysms in Kawasaki disease. Front Pediatr. 2021;9:630462.
32. Thangathurai J, Kalashnikova M, Takahashi M, Shinbane JS. Coronary artery aneurysm in Kawasaki disease: coronary CT angiography through the lens of pathophysiology and differential diagnosis. Radiol Cardiothorac Imaging. 2021;3:e200550.
33. Chan YH, Young CYM, Wang K, So ECT, Chu WCW. Giant coronary aneurysm in a toddler with Kawasaki disease: technical challenges in CT coronary angiography. BJR Case Rep. 2024;10:uaad008.
34. van Stijn D, Planken RN, Groenink M, Streekstra GJ, Kuijpers TW, Kuipers IM. Coronary artery assessment in Kawasaki disease with dual-source CT angiography to uncover vascular pathology. Eur Radiol. 2020;30:432-41.
35. Mavrogeni S, Papadopoulos G, Hussain T, Chiribiri A, Botnar R, Greil GF. The emerging role of cardiovascular magnetic resonance in the evaluation of Kawasaki disease. Int J Cardiovasc Imaging. 2013;29:1787-98.
36. Zheng X, Zhang Y, Liu L, Yue P, Wang C, Zhou K, et al. N-terminal pro-brain natriuretic peptide as a biomarker for predicting coronary artery lesion of Kawasaki disease. Sci Rep. 2020;10:5130.
37. Xu X, Wang J. Association between levels of N-terminal pro-brain natriuretic peptide and coronary artery lesion in patients with Kawasaki disease: a systematic review and meta-analysis. Arch Rheumatol. 2025;40:256-66.
38. Kim SH, Song ES, Yoon S, Eom GH, Kang G, Cho YK. Serum ferritin as a diagnostic biomarker for Kawasaki disease. Ann Lab Med. 2021;41:318-22.
39. Qiu Z, Liu HH, Fan GZ, Chen WX, Hu P. The clinical implications of serum ferritin in Kawasaki disease: a helpful biomarker for evaluating therapeutic responsiveness, coronary artery involvement and the tendency of macrophage activation syndrome. Arch Med Sci. 2022;18:267-74.
40. Wen H, Hun M, Zhao M, Han P, He Q. Serum ferritin as a crucial biomarker in the diagnosis and prognosis of intravenous immunoglobulin resistance and coronary artery lesions in Kawasaki disease: a systematic review and meta-analysis. Front Med. 2022;9:941739.
41. Brodeur KE, Liu M, Ibanez D, de Groot MJ, Chen L, Du Y, et al. Elevation of IL-17 cytokines distinguishes Kawasaki disease from other pediatric inflammatory disorders. Arthritis Rheumatol. 2024;76:285-92.
42. Lin IC, Suen JL, Huang SK, Chou MH, Kuo HC, Lo MH, et al. Involvement of IL-17 A/IL-17 receptor A with neutrophil recruitment and the severity of coronary arteritis in Kawasaki disease. J Clin Immunol 2024;44:77.
43. Huang YH, Kuo HC, Huang FC, Yu HR, Hsieh KS, Yang YL, et al. Hepcidin-induced iron deficiency is related to transient anemia and hypoferremia in Kawasaki disease patients. Int J

https://doi.org/10.59492/kd.2025.3.2.e12

Mol Sci. 2016;17:715.
44. Huang YH, Kuo HC. Anemia in Kawasaki disease: hepcidin as a potential biomarker. Int J Mol Sci. 2017;18:820.
45. Jone PN, Adriana Tremoulet C, Choueiter N, Dominguez SR, Harahsheh AS, Mitani Y, et al. Update on diagnosis and management of Kawasaki disease: a scientific statement from the American Heart Association. Circulation. 2024;150:e481-500.
46. Fukazawa R, Kobayashi J, Ayusawa M, Hamada H, Miura M, Mitani Y, et al. JCS/JSCS 2020 guideline on diagnosis and management of cardiovascular sequelae in Kawasaki disease. Circ J. 2020;84:1348-407.
47. Kobayashi T, Saji T,·Otani T, Takeuchi K, Nakamura T, Arakawa H, et al. Efficacy of immunoglobulin plus prednisolone for prevention of coronary artery abnormalities in severe Kawasaki disease (RAISE study): a randomised, open-label, blinded-endpoints trial. Lancet. 2012;379:1613-20.
48. Burns JC, Best BM, Mejias A, Mahony L, Fixler DE, Jafri HS, et al. Infliximab treatment of intravenous immunoglobulin–resistant Kawasaki disease. J Pediatr. 2008;153:833-8.E6.
49. Shafferman A, Birmingham JD, Cron RQ. High dose Anakinra for treatment of severe neonatal Kawasaki disease: a case report. Pediatr Rheumatol. 2014;12:26.
50. Tremoulet AH, Pancoast P, Franco A, Bujold M, Shimizu C, Onouchi Y, et al. Calcineurin inhibitor treatment of intravenous immunoglobulin–resistant Kawasaki disease. J Pediatr. 2012;161:506-12.E1.
51. Brogan P, Burns JC, Cornish J, Diwakar V, Eleftheriou D, Gordon JB, et al. Lifetime cardiovascular management of patients with previous Kawasaki disease. Heart. 2020;106:411-20.

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