Recurrent Acute Myocardial Infarction Associated with Coronary Artery Aneurysms Secondary to Kawasaki Disease in a Young Adult

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Introduction

Kawasaki disease (KD) is a systemic vasculitis that predominantly affects children younger than five years of age and is the leading cause of acquired heart disease in childhood.[1] Although uncommon in adults, its late cardiovascular complications include coronary artery aneurysms, which may lead to acute myocardial infarction (AMI), sudden cardiac death, or heart failure.[2] Evidence regarding the management of adults with cardiac sequelae of Kawasaki disease (KD) remains limited, highlighting the importance of case reports.

Initial presentation

A 23-year-old man of East Asian ancestry with a history of untreated Kawasaki disease (KD) during childhood presented with a late anterior ST-segment elevation myocardial infarction (STEMI) (Figure 2). Coronary angiography (Figure 3) revealed a giant aneurysm involving the entire left main coronary artery (LMCA) and the proximal third of the left anterior descending artery (LAD), in addition to a 40% stenosis in the mid-distal LAD and a 50% stenosis in the proximal right coronary artery (RCA), with alternating stenotic and ectatic segments. Transthoracic echocardiography demonstrated akinesis of the mid inferoseptal, mid anteroseptal, and apical septal segments, with a left ventricular ejection fraction of 39% measured by the Simpson biplane method. Whole-body computed tomography angiography showed no evidence of aneurysms in other arterial territories. The patient was discharged on ramipril, metoprolol, rosuvastatin, aspirin, clopidogrel, and anticoagulation with a vitamin K antagonist (VKA), which was subsequently replaced with rivaroxaban because of poor treatment adherence.

Initial follow-up

One year later, myocardial perfusion scintigraphy (MPS) demonstrated persistent moderate hypoperfusion in the anterior, anteroseptal, and apical walls, with mild transient hypoperfusion in the anterior wall, corresponding to a post-stress perfusion defect involving 17% of the left ventricle and a resting perfusion defect of 16% (Figure 4A). Two years later, repeat MPS demonstrated improvement, with a reduction in the perfusion defect to 10% (Figure 4B).

Recurrence three years later

At 26 years of age, the patient experienced a recurrent acute myocardial infarction after inappropriate discontinuation of oral anticoagulation for three days. Coronary angiography demonstrated persistence of the LMCA aneurysm, thrombotic occlusion of the LAD at its origin, and a high thrombus burden. Mechanical aspiration thrombectomy followed by intracoronary tirofiban administration was performed, restoring TIMI 3 flow despite residual thrombus burden (Figures 5A and 5B). On the fourth day of hospitalization, the patient developed a recurrent coronary event with no significant angiographic changes compared with the initial findings, and conservative management was therefore adopted. Transthoracic echocardiography showed mildly reduced left ventricular systolic function, with an ejection fraction of 45% and no new regional wall motion abnormalities.

The patient had an uneventful clinical course and was discharged on ticagrelor, rivaroxaban, rosuvastatin, ramipril, and metoprolol. Five months after discharge, left ventricular ejection fraction improved to 50%. Eight months later, he was asymptomatic, with a new MPS demonstrated persistent hypoperfusion of the anteroseptal wall and mild hypoperfusion of the inferoapical wall, with no stress-induced ischemia and a fixed perfusion defect involving 26% of the left ventricle (Figure 4C). At the most recent follow-up, the patient remained in New York Heart Association (NYHA) functional class I, with no recurrent coronary events, as summarized in the timeline shown in Figure 1.

Discussão

In untreated Kawasaki disease (KD), coronary artery aneurysms develop in approximately 15% to 25% of patients and represent the most serious cardiovascular complication of the disease, potentially leading to acute myocardial infarction, sudden cardiac death, or heart failure.[3] Prognosis is primarily determined by the initial extent of coronary artery involvement, with certain patient subgroups being at higher risk of developing myocardial ischemia secondary to coronary thrombosis or stenosis resulting from the underlying inflammatory process.[4]

Keywords

Mucocutaneous Lymph Node Syndrome; Coronary Aneurysm; Myocardial Infarction; Anticoagulants

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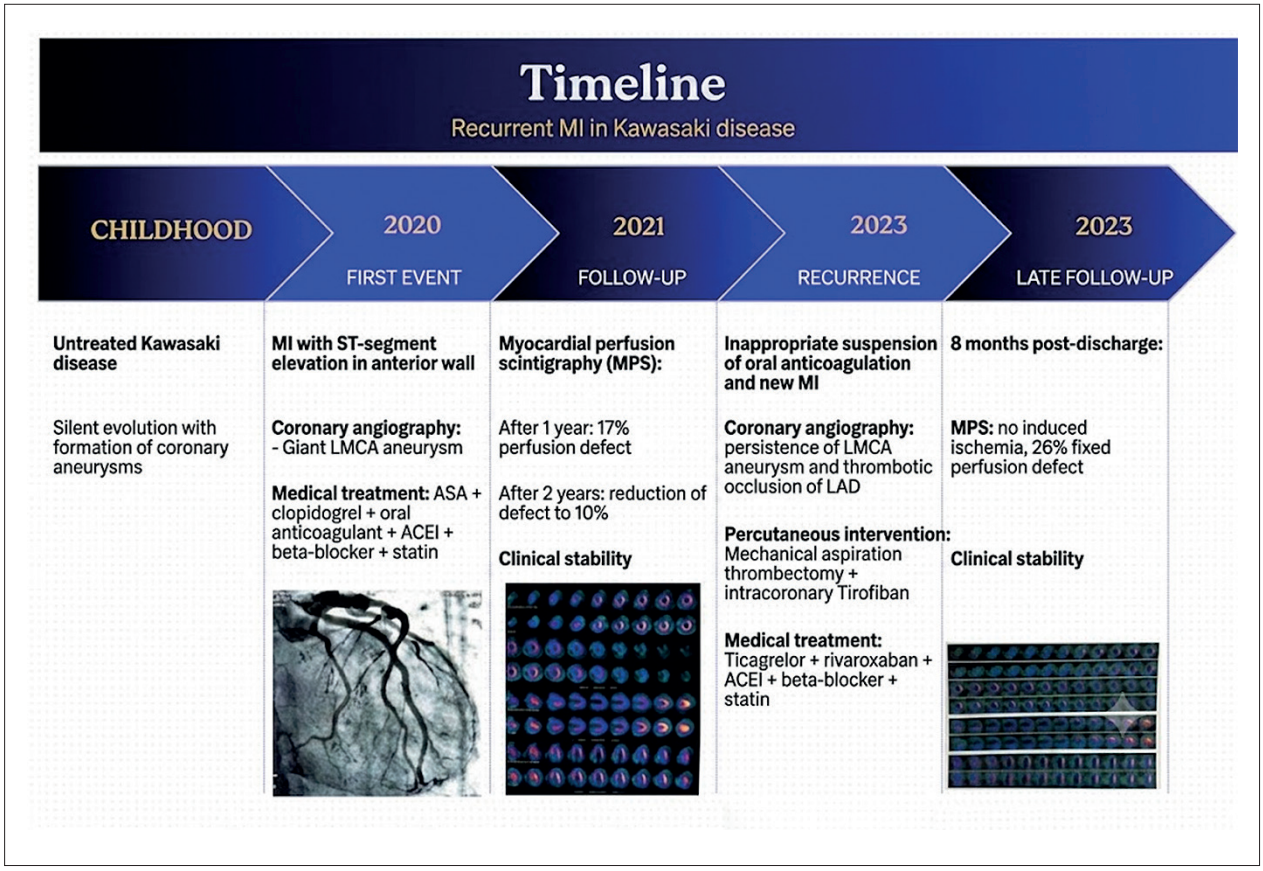


Figure 1 – *Timeline. Chronology of the patient's clinical presentation and progression. MI: myocardial infarction; LMCA: left main coronary artery; ACEI: angiotensin-converting enzyme inhibitor; LAD: left anterior descending artery.*

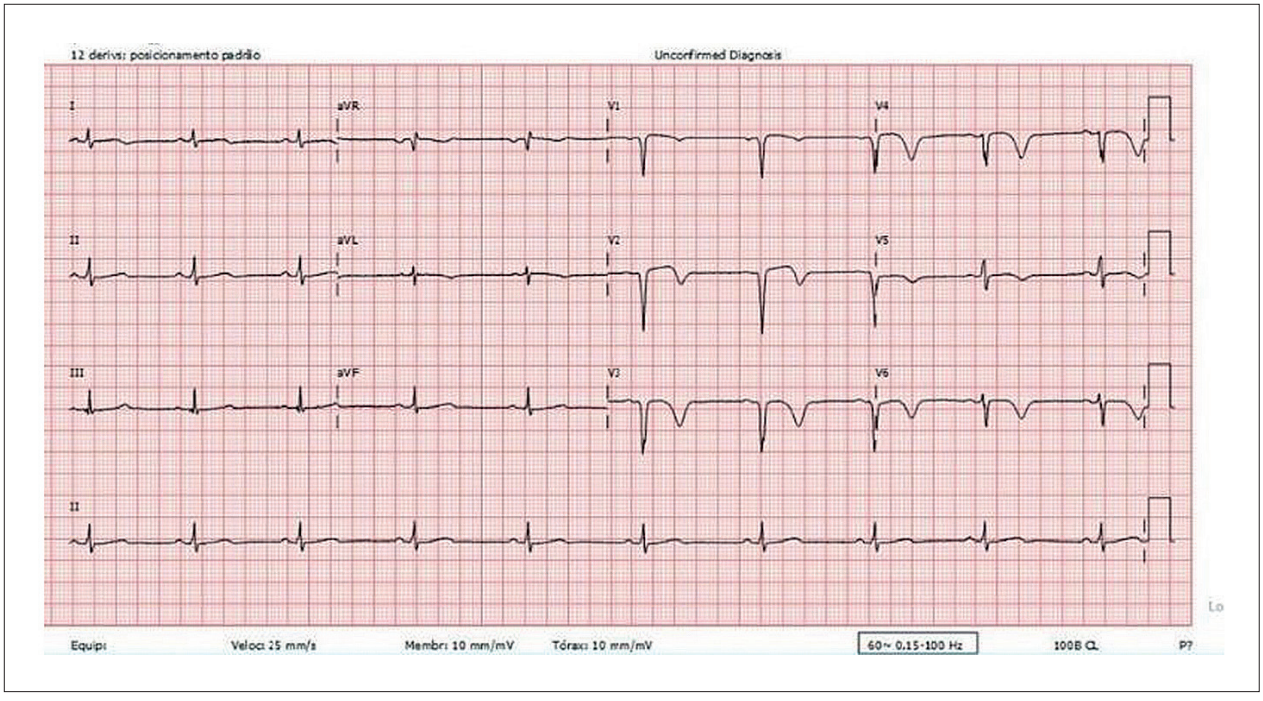


Figure 2 – *Electrocardiogram showing typical ischemic changes.*

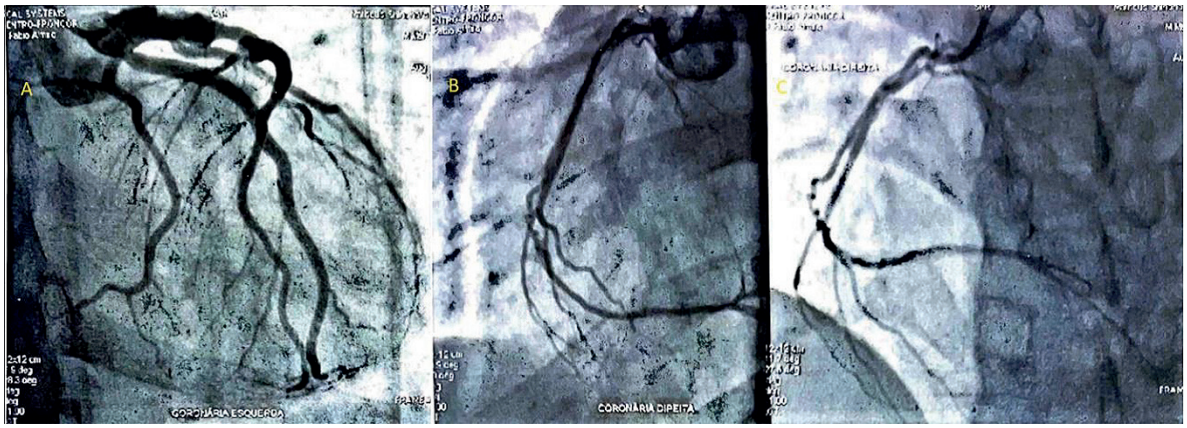


Figure 3 – *Coronary angiography showing a left main coronary artery (LMCA) aneurysm.*

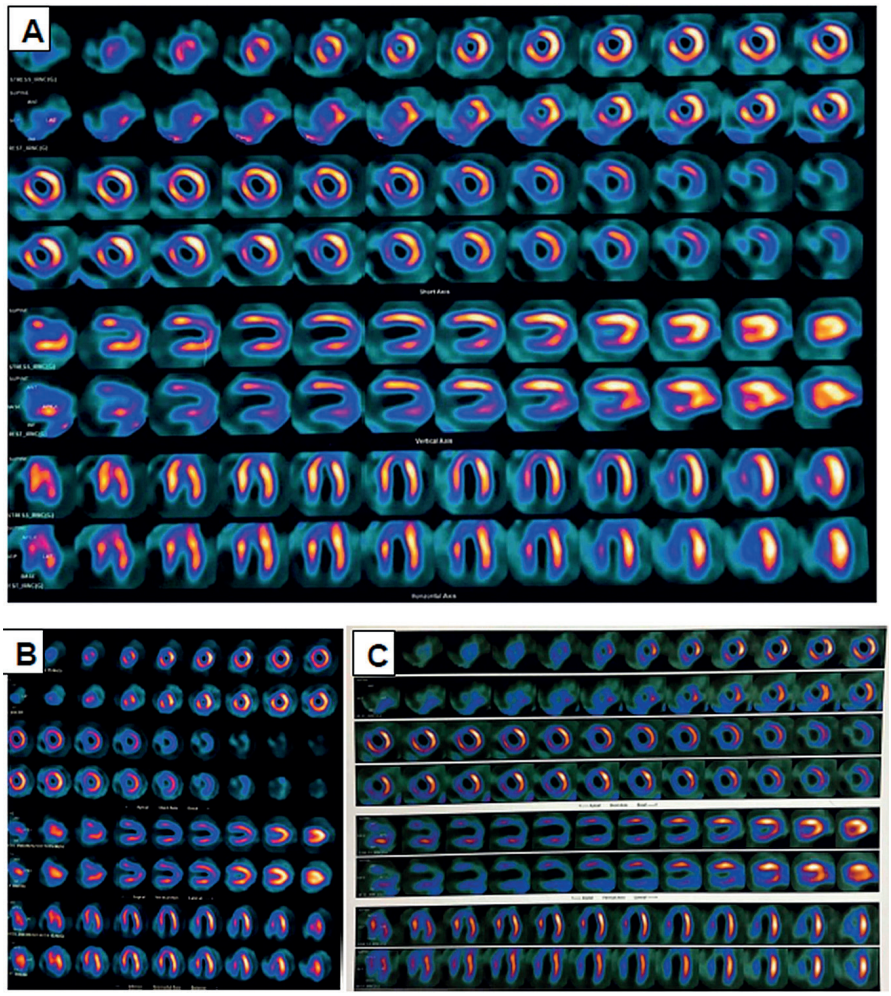


Figure 4 – *A) Myocardial perfusion scintigraphy (MPS) one year after the initial event. B) MPS two years after the initial event. C) Follow-up MPS eight months after the recurrent event.*

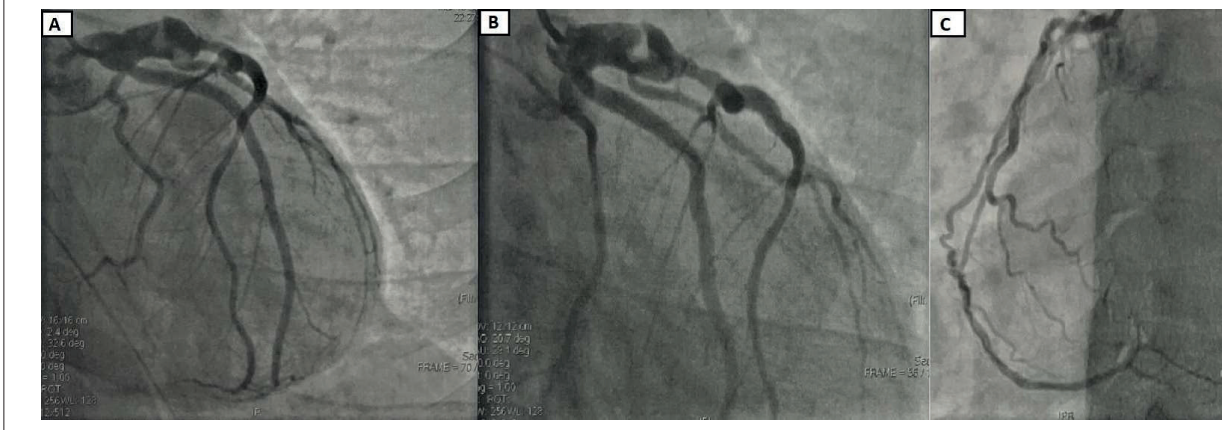


Figure 5 – *A) Coronary angiography demonstrating a left main coronary artery (LMCA) aneurysm with a high thrombus burden. B) Patent left anterior descending artery (LAD) following mechanical thrombectomy, demonstrating a myocardial bridge in the mid segment. C) Right posterior descending artery and right posterior ventricular artery showing a corkscrew-like vascular configuration.*

The inflammatory response, mediated by cytokines such as interleukin (IL)-1, IL-6, and tumor necrosis factor-alpha (TNF-α), causes injury and weakening of the arterial wall, leading to aneurysm formation and, consequently, reduced blood flow, low shear stress, blood stasis, and intraluminal thrombus formation. During the chronic phase, myofibroblastic proliferation promotes progressive coronary stenosis.[5]

The association between chronic inflammatory rheumatic diseases and cardiovascular disease has been attributed, at least in part, to inflammation-mediated endothelial dysfunction. Angiotensin-converting enzyme (ACE) inhibitors exert both hemodynamic and anti-inflammatory effects by inhibiting angiotensin II production,[6] supporting their initial use in this patient because of left ventricular dysfunction.

Among the few similar cases reported, one involved a 69-year-old man with a history of childhood Kawasaki disease who presented with acute myocardial infarction associated with aneurysmal occlusion of the RCA and multiple fusiform coronary artery aneurysms. He was managed medically with alteplase on admission, followed by prolonged dual antiplatelet therapy, because percutaneous coronary intervention was not feasible. This case highlights the limitations of percutaneous treatment in Kawasaki vasculopathy due to extensive stenotic and aneurysmal coronary lesions.[7]

Although VKAs remain the preferred anticoagulant therapy for patients with giant coronary artery aneurysms secondary to Kawasaki disease,[2] direct oral anticoagulants (DOACs), particularly rivaroxaban, have emerged as an alternative when warfarin therapy is impractical, such as in patients with poor adherence to international normalized ratio (INR) monitoring.[8] In the present case, replacing warfarin with rivaroxaban was considered clinically justifiable because long-term VKA therapy could not be maintained.

On the other hand, combining a DOAC with ticagrelor should be interpreted with caution, as evidence supporting this strategy in Kawasaki disease remains limited and may be associated with an increased risk of bleeding. Therefore, treatment was individualized based on the patient's high thrombotic risk, multivessel coronary artery disease, left ventricular dysfunction, prior myocardial infarction at a young age, and the need to maximize adherence to anticoagulant therapy.

Emerging evidence also suggests that factor Xa inhibition may represent a promising strategy for preventing thrombotic events in adults with cardiovascular sequelae of Kawasaki disease.[9]

Currently, there is no established consensus regarding the management of cardiovascular complications of Kawasaki disease in adulthood, particularly when associated with coronary artery disease and heart failure. Antiplatelet therapy, anticoagulation according to aneurysm size, angiotensin-converting enzyme (ACE) inhibitors, and statins remain the main therapeutic strategies.[8]

This case illustrates that, in patients with Kawasaki disease and giant coronary artery aneurysms, antithrombotic therapy should balance thrombotic and bleeding risks. In the absence of prospective studies in adults, therapeutic decisions remain individualized and guided by current recommendations. Case reports such as this may help guide the management of similarly complex cases until more robust evidence becomes available.

Conclusion

Recurrent acute myocardial infarction in adults with sequelae of untreated Kawasaki disease represents a therapeutic challenge. Individualization of management strategies and strict adherence to antithrombotic therapy are crucial to prevent recurrences and preserve ventricular function.

Author Contributions

Conception and design of the research and Critical revision of the manuscript for important intellectual content: Silva

Junior DG; Data collection: Silva Junior DG, Pereira Filho EG; Data analysis and interpretation, Statistical analysis and Manuscript drafting: Silva Junior DG, Pressuto KMA, Bottaro KIPD, Lepre VBN.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Hospital Universitário Maria Aparecida Pedrossian under the protocol number 7454560. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Data Availability Statement

All datasets supporting the results of this study are available upon request from the corresponding author.

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