A Challenging Case of Kawasaki Disease with Systemic Vasculopathy and Graft Failure Soon After CABG, Presenting with Persistent Chest Pain

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Abstract

Managing late cardiovascular sequelae of Kawasaki disease (KD) in adults poses significant challenges in interventional cardiology. We present a case of KD with systemic vasculopathy and complex triple-vessel coronary artery disease (CAD) with long treatment course. In 2019, angiography revealed CAD with a SYNTAX score of 62 points. Consequently, coronary artery bypass grafting (CABG) was performed. Three months later, the patient presented with recurrent chest pain and follow-up angiography revealed complete loss of the grafts to the right coronary artery (RCA) and left circumflex artery (LCX), despite patent left internal mammary artery (LIMA) graft. Lower limb angiography indicated diffuse and small bilateral common femoral and iliac arteries. The patient underwent four challenging percutaneous coronary intervention (PCI) procedures, including rotablation of the native left anterior descending artery (LAD) and retrograde PCI to open a difficult LCX chronic total occlusion (CTO) via the LAD. Addressing the systemic vasculopathy, the patient was diagnosed with Sjogren's syndrome and treated with hydroxychloroquine. Follow-up showed significant improvement in femoral and iliac vessel sizes. This case underscores the need for multidisciplinary management and advanced treatment approaches for complex KD-related coronary lesions.

Keywords: Kawasaki disease, chronic total occlusions, rotational atherectomy, ischemic heart disease

Introduction

Kawasaki disease (KD) is a form of vasculitis that can result in systemic vasculopathy and advanced coronary lesions. The latter include coronary aneurysms of various sizes, often heavily calcified and thrombotically occluded, and tight and complex coronary stenosis due to fibrotic changes in the endothelial and media layers, frequently heavily calcified as well.[1] Managing

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these late and complex coronary lesions in adults poses significant challenges for cardiologists. Lesions that are too complex and calcified may exceed the scope of percutaneous interventions and can only be managed with supportive treatment. Even less complex lesions, such as chronic total occlusion (CTO), thrombotically occluded aneurysms, and tight stenosis, can also present considerable challenges as they are often heavily calcified. A tailored treatment plan, along with an advanced and adaptable intervention toolbox, remains crucial for addressing these complex coronary lesions.

Patients diagnosed with conditions like KD and Sjögren's syndrome might develop a type of vasculitis affecting medium-sized arteries, similar to what is observed in polyarteritis.[2] Treatment for systemic vasculitis in Sjögren's syndrome typically entails a multifaceted approach, including medications like hydroxychloroquine, oral glucocorticoids, oral Disease-Modifying Antirheumatic Drugs (DMARDs) such as methotrexate and leflunomide, along with biological therapies like rituximab.[3]

Herein, we present a case of adult KD with systemic vasculopathy related to KD and Sjögren's syndrome, and complex triple-vessel coronary disease. Due to the complexity of the coronary lesions, the patient underwent a total of four challenging percutaneous coronary intervention (PCI) procedures, including rotablation to address the heavily calcified left anterior descending artery (LAD) (3rd PCI, complicated by slow flow and cardiogenic shock) and retrograde PCI via the LAD to finally open the occluded left circumflex artery (LCX) (4th PCI). Addressing the systemic vasculopathy, the patient was diagnosed with Sjogren's syndrome and treated with hydroxychloroquine. This case report underscores the importance of multidisciplinary management of KD with systemic vasculopathy and the need for patience and advanced treatment approaches to complex coronary lesions in such patients.

Case Report

A 41-year-old female presented with non-ST-elevation myocardial infarction (NSTEMI) to the local hospital and was diagnosed with complex triple-vessel coronary disease with a SYNTAX score of 62 points, based on coronary angiography (CAG). Following stabilization of her hemodynamics by intra-aortic balloon pump (IABP), she underwent bypass surgery uneventfully. The left internal mammary artery (LIMA) was anastomosed to the LAD, and saphenous vein grafts (SVGs) were used for both the posterior descending artery (PDA) and LCX. Nevertheless, the patient suffered a recurrence of the chest pain just three months after the bypass surgery and sought medical help from our hospital. Echocardiography revealed generalized left ventricular hypokinesis with regional wall motion abnormalities and a left ventricular ejection fraction of 30%. A myocardial perfusion imaging study showed marked reversible decrease in perfusion in the mid to basal anterior wall, and a partially reversible decrease in myocardial perfusion in the apical to basal lateral wall and the apical to basal inferior wall (Figure 1). The patient was receiving guideline-directed medical therapy for heart failure, which included medications such as angiotensin receptor neprilysin inhibitor, beta blocker, mineralocorticoid receptor antagonist, and sodium-glucose cotransporter-2 inhibitor.

Follow-up CAG revealed a small heavily-calcified aneurysm at the distal left main artery (LM) and another small aneurysm at the proximal-middle junction of the LAD surrounded by circular heavily-calcified 95% segmental stenoses at both flanks (Figure 2A). The LCX was completely occluded at the middle portion with a tiny lumen beyond the CTO segment, while the distal vessel bed was poorly visualized due to underdeveloped collaterals (Figure 2B). The right coronary artery (RCA) was entirely occluded from the ostium to the crus, with the PDA and posterolateral (PL) branches supplied by a large atrial collateral from

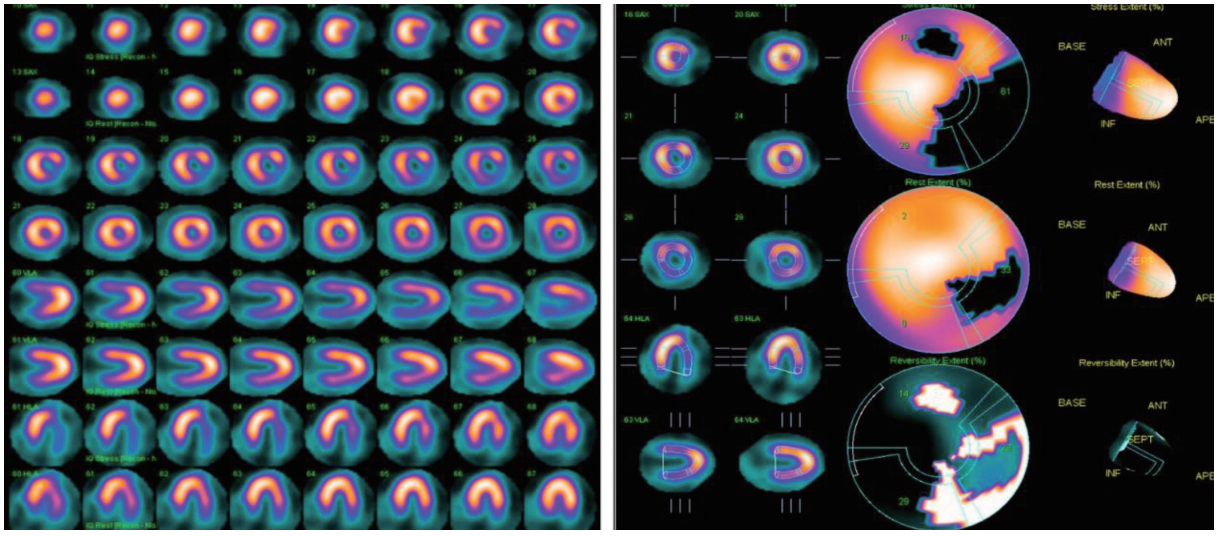

Figure 1. A myocardial perfusion imaging study revealed a significant reversible decrease in perfusion in the mid to basal anterior wall, as well as a partially reversible decrease in myocardial perfusion in the apical to basal lateral wall and the apical to basal inferior wall.

the mid-LCX and septal collaterals (Figure 2D). Notably, the LIMA graft remained patent and supplied the LAD-D (Figure 2C), whereas the SVGs to both the PDA and LCX were completely gone.

During the first PCI attempt, the mid-LCX CTO was sequentially probed with Gaia First and Fielder XT-A wire (Asahi Intecc Medical), while supported by a Finecross microcatheter (Terumo Corporation). However, the wire's position in the distal vessel bed could not be ascertained as the distal vessel bed could not be visualized clearly due to lack of antegrade blood flow and very poor collaterals (Figure 2E). After the CTO segment was sequentially dilated with 1.0 x 6 mm, 1.5 x 20 mm, and 2.0 x 20 mm balloons, the attempt had to be halted, despite the antegrade blood flow being only Thrombolysis in Myocardial Infarction (TIMI) grade 2 (Figure 2F). The femoral angiogram prior to the intended angiosealing of vascular access revealed diffuse small-sized femoral and iliac arteries on both sides, completely precluding the feasibility of angiosealing (Figure 2G-H). Due to strong evidence of systemic vasculopathy, including poor coronary collateral formation, diffuse small-sized femoral and iliac arteries on both sides and rapidly occluded SVGs, the patient was referred to our rheumatologist. In the course of a thorough examination, the patient presented with positive SSA antibodies, indicative of autoimmune involvement. Clinical findings included decreased tear production (Schirmer test), reduced tear meniscus, and sialoscintigraphy showing impaired salivary gland function. These findings, along with the presence of anti-SSA antibodies and characteristic ocular and salivary manifestations, supported the diagnosis of Sjögren's syndrome. Systemic vasculopathy was deemed to be a part of both Sjögren's syndrome and Kawasaki disease; therefore, hydroxychloroquine daily was prescribed.

A second attempt (2nd time) at PCI for the LCX CTO was made two months later via right femoral access. Despite prolonged maneuvering, successful wiring of the LCX CTO was not achieved, using Fielder XT-R, Fielder XT-A, or Conquest Pro wires (all by Asahi Intecc Medical), supported by a Finecross microcatheter (Terumo Corporation), due to the existing dissections

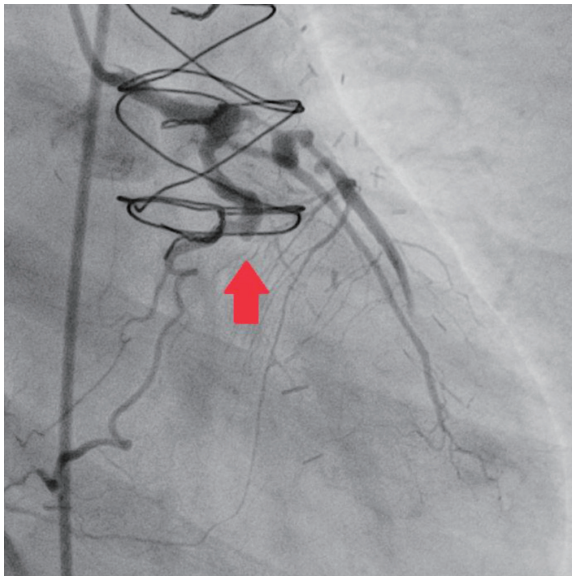

Figure 2A. Diagnostic coronary angiography. The LCX was completely occluded at the middle portion with a tiny lumen beyond the CTO segment (arrow), while the distal vessel bed was poorly visualized due to underdeveloped collaterals.

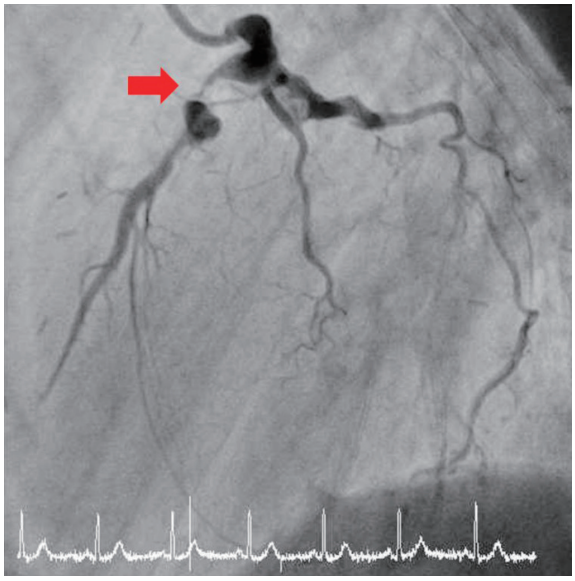

Figure 2B. Diagnostic coronary angiography. A small heavily-calcified aneurysm at the distal left main artery and another small aneurysm at the proximal-middle junction of the LAD surrounded by circular, heavily-calcified 95% segmental stenoses (arrow).

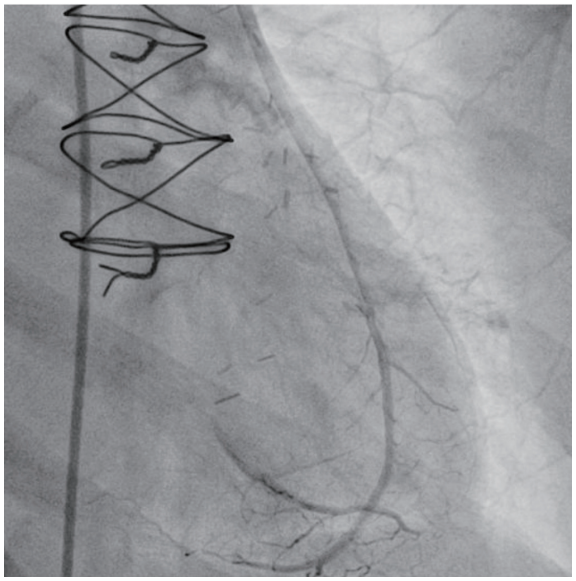

Figure 2C. Diagnostic coronary angiography. The LIMA graft remained patent and supplied the LAD-D.

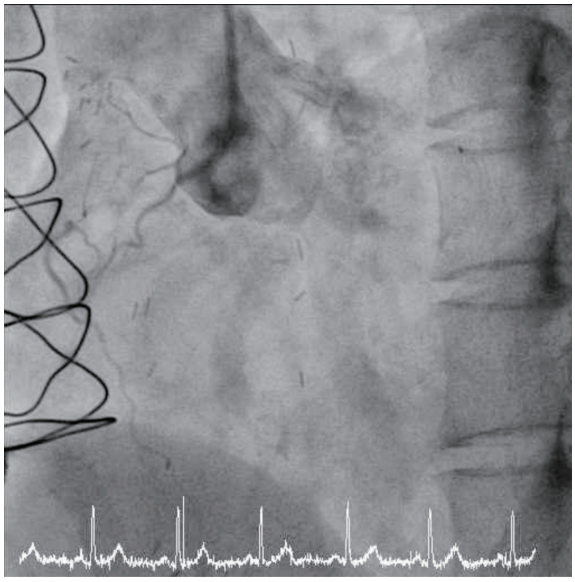

Figure 2D. Diagnostic coronary angiography. The RCA is likely a non-dominant vessel, and there is a very long CTO extending from the ostium of the RCA.

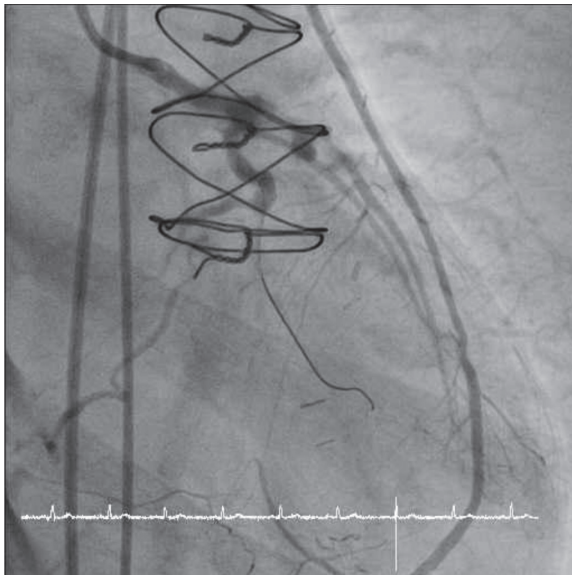

Figure 2E. The Gaia first guidewire (Asahi Intecc Medical) managed to navigate the CTO segment with the assistance of a Finecross microcatheter (Terumo Corporation).

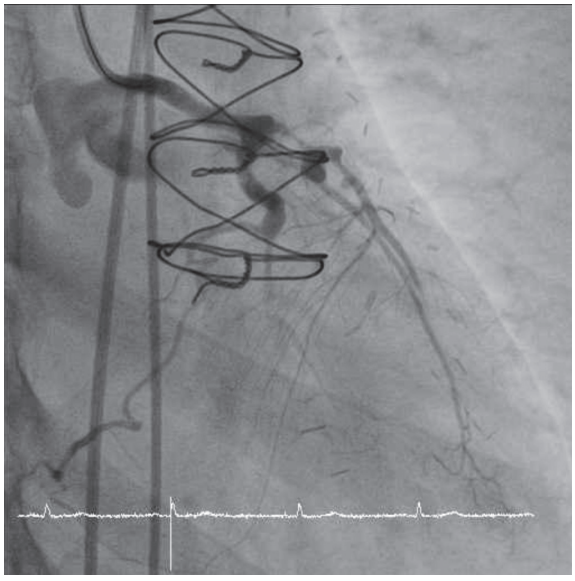

Figure 2F. Final angiography showed the upper branch of OM1 distal to the CTO was dilated with antegrade blood flow.

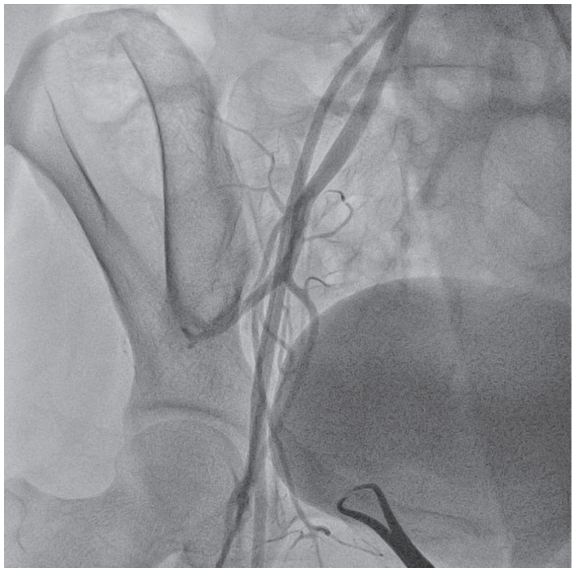

Figure 2G. The femoral angiogram conducted before the intended angiosealing of vascular access showed bilateral diffuse small-sized femoral and iliac arteries.

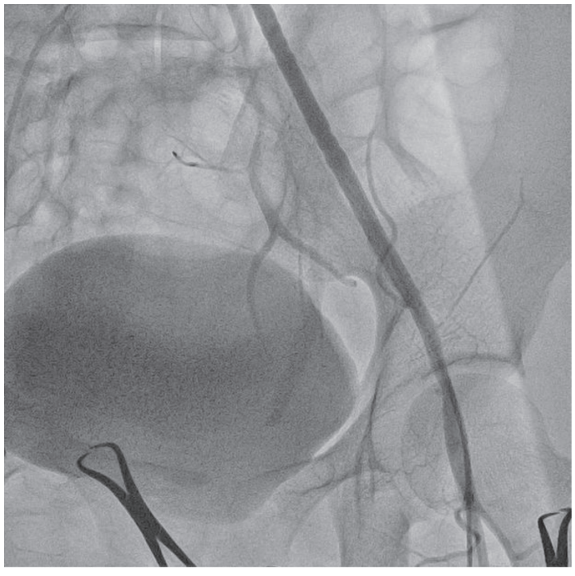

Figure 2H. The femoral angiogram conducted before the intended angiosealing of vascular access showed bilateral diffuse small-sized femoral and iliac arteries.

left over from the previous PCI attempts and the unfavorable anatomies seen in the previous intervention. Therefore, the procedure had to be abandoned to avoid causing more dissections. The patient continued with dual-antiplatelet therapy for six months, followed by clopidogrel monotherapy, in addition to anti-ischemic drugs including a beta-blocker, calcium channel blocker, and nitrate. Unfortunately, she still suffered from severe chest pain in the meantime. Due to the poor coronary anatomy and the alleged limited ischemic territory in the LCX area based on the previous coronary angiogram, only supportive care was offered. Due to the patient's emotional vulnerability and strong affective reactions to chest pain, she was suspected to have hysteria and not true coronary ischemia. The patient was referred to a psychiatric outpatient clinic for chest pain management.

Three years later, the patient finally underwent a CAG follow-up due to very disturbing chest pain and poor quality of life. This time, the LIMA and LAD were found to have remained the same, but the LCX occlusion had extended retrogradely all the way to the ostium, resulting in a stumpless CTO starting from the distal LM aneurysm (Figure 3A). Furthermore, the large atrial collateral from the mid-LCX to RCA-PL was completely lost (Figure 3B). We concluded that the patient's chest pain was caused by progressive ischemia in the LCX and RCA-PL territories and not by psychological issues. Due to the stumplessness, retrograde PCI (3rd PCI) via the LAD to open the LCX CTO was intended, while the LIMA was never considered as the antegrade limb as this vessel was the lifeline for LAD blood flow. However, the Caravel microcatheter (Asahi Intecc Medical) failed to pass through the heavily calcified critical stenoses of the proximal-middle LAD, requiring rotational atherectomy of the LAD before retrograde PCI could proceed. After switching to the extra-support Rota wire (Boston Scientific), the LAD-P-M lesions were slowly debulked with a 1.5 mm burr (Figure 3C), running a few times but then quickly followed by severe systemic hypotension. Emergent angiographic check of the LIMA found no occlusion, but the flow to the LAD and septal branches became very much compromised. We concluded that the acute slow flow phenomenon complicating rotablation caused compromised flow to the PDA and PL territory and was the likely cause for the acute hemodynamic event. After emergency measures, the hemodynamics were stabilized, but further attempts were halted.

Another attempt (4th PCI) to complete the retrograde approach was undertaken three months later. This time, the microcatheter went through the LAD-P-M and down the LAD without any resistance (Figure 3D-E). The septal collateral to the distal LCX was successfully navigated with a Fielder-XTR (Asahi Intecc Medical), and a Corsair Pro XS (Asahi Intecc Medical) was advanced all the way to the distal LCX, despite the collateral being tiny and bent. Due to the long and heavily calcified CTO body, undetermined CTO route, lack of options for an antegrade approach due to stumplessness, and the distal LM aneurysm, only retrograde wiring was possible, albeit very difficult (Figure 3F). This was finally accomplished with a Conquest Pro 12 (Asahi Intecc Medical) after a long struggle. After RG 3 wire (Asahi Intecc Medical) externalization, the LCX CTO was balloon dilated and scaffolded with a DES, resulting in excellent final flow on angiography (Figure 3G). At the end of the procedure, follow-up showed significant improvement in femoral and iliac vessel sizes on both sides after years of regular hydroxychloroquine treatment (Figure 2H). During outpatient follow-up visits, the patient experienced no further chest pain. Remarkably, she was able to participate in a family outing, marking a significant improvement in her quality of life.

Discussion

Kawasaki disease (KD), primarily seen in children, poses significant challenges in adult

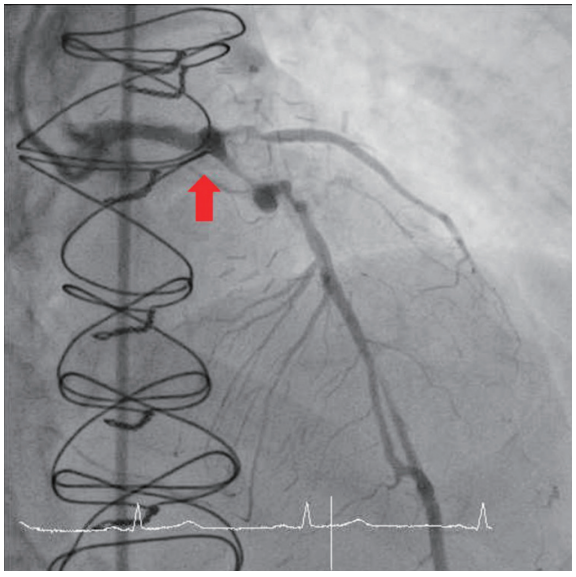

Figure 3A. The LCX occlusion extended retrogradely to the ostium, leading to a stumpless CTO originating from the distal LM aneurysm (arrow). Additionally, the significant atrial collateral from the mid-LCX to RCA-PL was completely lost.

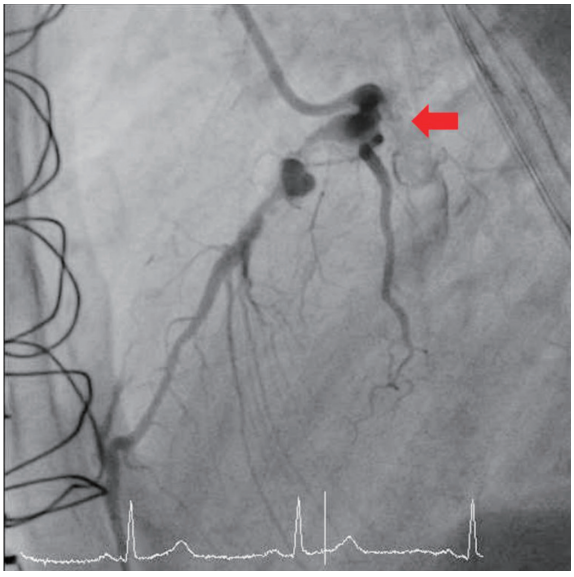

Figure 3B. The LCX occlusion extended retrogradely to the ostium, leading to a stumpless CTO originating from the distal LM aneurysm (arrow). Additionally, the significant atrial collateral from the mid-LCX to RCA-PL was completely lost.

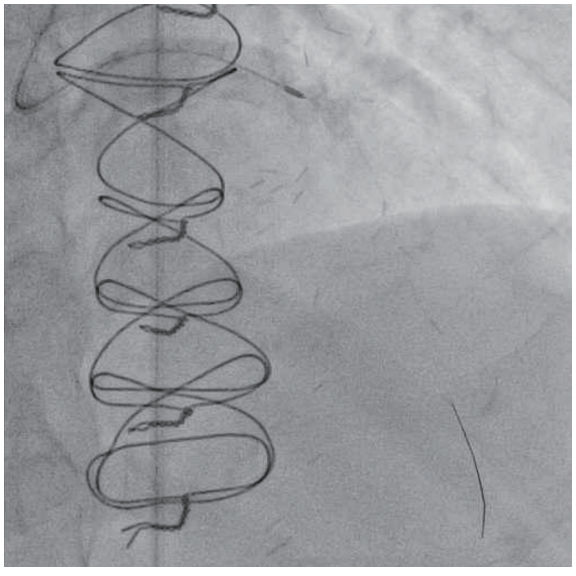

Figure 3C. The lesions in the LAD were treated with rotational atherectomy.

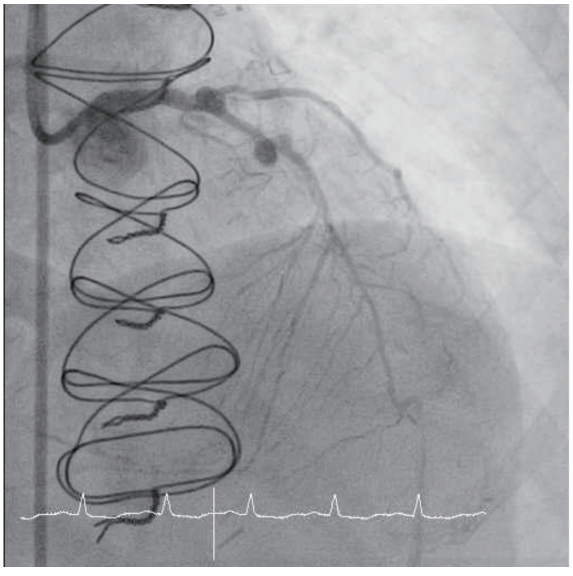

Figure 3D. A very good final flow was observed in the native LAD after successful rotational atherectomy.

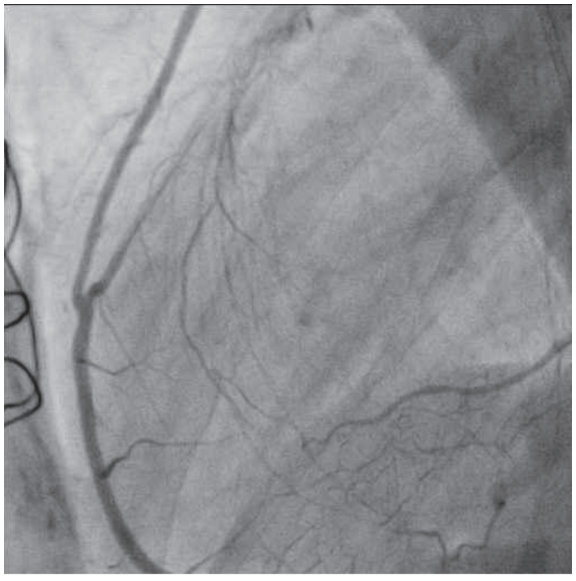

Figure 3E. Contralateral injection via the LIMA graft showed prominent collaterals from the LAD to the LCX.

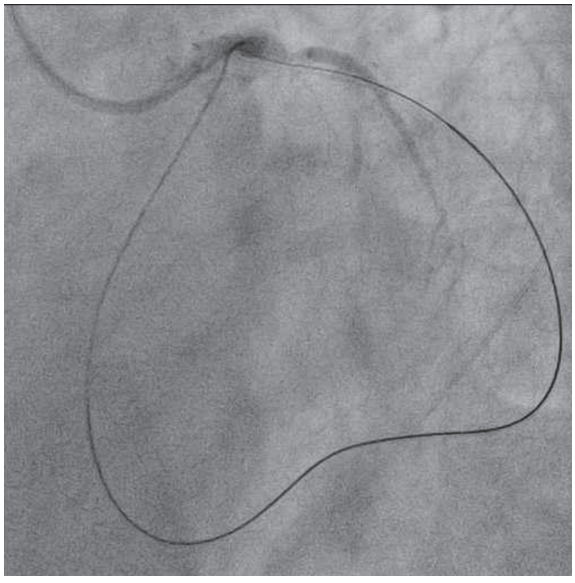

Figure 3F. The retrograde wiring inadvertently crossed the CTO cap at the last moment.

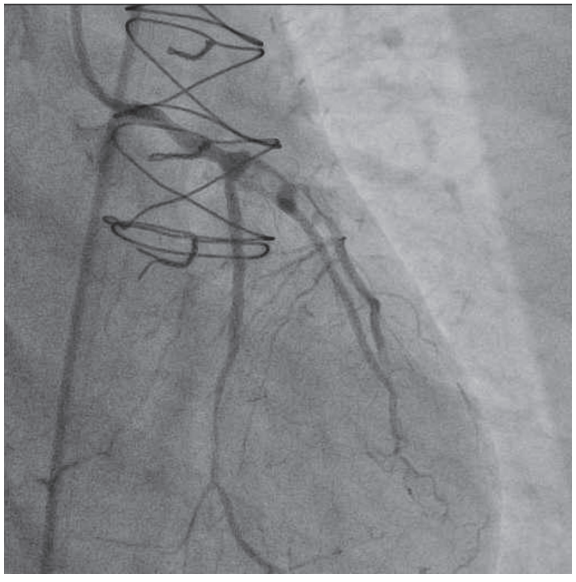

Figure 3G. The final angiography revealed a successful restoration of blood flow from the LCX.

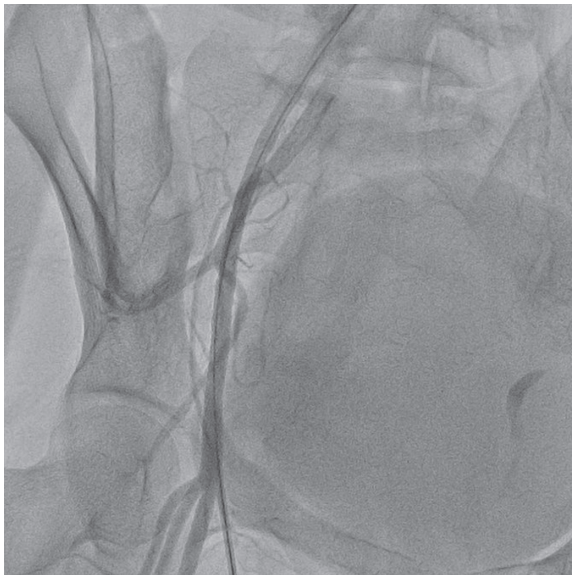

Figure 3H. The follow-up revealed a substantial improvement in the sizes of the femoral and iliac vessels.

cardiology, especially when patients develop severe cardiovascular issues such as coronary artery aneurysms which are often thrombotically occluded, and critical stenoses which are mostly heavily calcified. Managing these complications through PCI requires dedicated strategies, advanced skills and a powerful toolbox to ensure effective treatment and patient safety. The coronary lesions in KD exhibit a distinct pathophysiology compared to coronary atherosclerosis. One of the major challenges is the presence of coronary artery aneurysms. The aneurysmal segments are frequently associated with increased tortuosity, heavy calcification, myointimal thickening, and thrombotic occlusion.[4] Moreover, patients with aneurysms face a high probability of coronary artery calcification development. Overall, significant calcification usually takes up to ten years to manifest.[5] This calcification could be the eventual outcome of the initial inflammatory insult linked to coronary aneurysm formation. Therefore, the complexities of coronary artery disease (CAD) in KD are often profound and may involve all three vessels, and include coronary artery aneurysms of different sizes, vessel fibrosis, and significant calcifications which are often thick and circular.[6] These features profoundly complicate any PCI procedure. Intravascular ultrasound (IVUS) is particularly valuable in this context, offering detailed qualitative and quantitative data on lesion calcification and aneurysm characteristics, and aiding in procedural planning. Optical coherence tomography (OCT) can provide better image quality in heavily calcified lesions, but it is not useful for aneurysmal lesions due to its imaging depth.[7] However, heavily calcified lesions often impede the use of intra-coronary imaging devices and even microcatheters of very small crossing profile, as observed in our case.

During the intervention phase, the traditional approach such as plain old balloon angioplasty (POBA) often yields suboptimal results due to the rigidity of calcified lesions.[8] Alternative balloons like cutting and scoring balloons are sometimes employed to achieve adequate lesion modification.[9] However, thick calcification often does not respond. High-pressure balloon dilation can be effective for heavily calcified lesions but raises concerns regarding the creation of neoaneurysms.[8,10] In our experience, the most heavily calcified coronary lesions in KD patients demand rotational atherectomy. Rotational atherectomy is currently recommended by guidelines for the management of calcified lesions in patients with Kawasaki disease.[6,11] Sometimes, vessels remain undilatable even after rotablation. Therefore, experts recommend the use of larger burrs, generally greater than 2.15 mm in diameter, to achieve better long-term patency results.[6,12] However, this technique requires meticulous procedural handling and could be associated with slow flow or no-reflow phenomena, especially in the presence of residual thrombosis within aneurysms. This was also seen in our patient after LAD rotablation. Passing the rotational burr through an aneurysm demands high precision to avoid complications like coronary perforation.[13] A novel and promising approach is the use of shockwave intravascular lithotripsy (IVL) to treat calcified lesions in KD patients.[14] This technique employs acoustic shockwaves to fracture calcified plaques within the vessel walls without causing direct injury to the vessel itself. IVL's mechanism reduces the risk of distal embolization and other complications associated with traditional balloon and atherectomy methods.[15] Although this approach shows potential for enhanced safety outcomes, further long-term efficacy data in KD patients are still required to validate its widespread application. Regardless of technique employed, follow-up angiography remains crucial for all these patients. Previous studies have shown that if a target vessel remains patent one year after rotational atherectomy, it is likely to maintain long-term patency.[16]

In addition to the treatment toolbox, advanced intervention skills with experience

and expertise are often required to treat these coronary lesions, to provide detailed film reading before procedures, perform antegrade and/or retrograde approaches for complex and advanced CTO lesions, and demonstrate perseverance in percutaneous treatment, as seen in this patient. According to the literature, aneurysms were already found in a study during the intravenous immunoglobulin (IVIG) therapy era. Persistent coronary artery aneurysms (CAAs) are notably less prevalent, affecting 4% to 6% of patients, with approximately 1% progressing to giant CAAs, defined as dilatation exceeding 8 mm.[17] Giant CAAs are significantly linked to unfavorable patient outcomes, given that the increasing size of these aneurysms heightens the risk of thrombosis, rupture, stenosis, myocardial infarction, and sudden death.[18] The thrombophilic risk in patients with KD was well demonstrated in this patient, as evidenced by the occlusion of the LCX-P during the three-year follow-up period, leading to compromised collateral blood flow in the LCX and RCA-PL territory and worsening chest pain. Unfortunately, due to the complex clinical presentations, the patient was suspected to have some psychological issues. Fortunately, the angiographic follow-up revealed the cause of the problem, and the successful opening of the CTO lesions provided relief. Despite the decreased occurrence of CAAs with IVIG treatment, CAAs resulting from Kawasaki disease in childhood represent 5% of acute coronary syndromes (ACS) in adults below 40 years of age.[19] However, there have only been a few case reports regarding patients with Kawasaki disease developing CTO. Further investigation in this area is necessary.

Patients diagnosed with conditions like Kawasaki disease (KD) and Sjögren's syndrome might develop a type of vasculitis affecting medium-sized arteries, similar to what is observed in polyarteritis.[2] Previous studies have shown that children with KD have increased long-term risks of not only ischemic heart disease but also autoimmune diseases.[20] Therefore, a multidisciplinary evaluation is mandatory to ensure an accurate diagnosis. Generally, the management of systemic vasculitis in Sjögren's syndrome involves the use of hydroxychloroquine, oral glucocorticoids, oral Disease-Modifying Antirheumatic Drugs (DMARDs) such as methotrexate, leflunomide, and azathioprine, as well as biological therapies like rituximab, abatacept, and belimumab. Treatment typically starts with daily hydroxychloroquine, followed by low-dose weekly methotrexate or oral glucocorticoids. In a small percentage of cases, rituximab, mycophenolate mofetil, abatacept, azathioprine, and belimumab may be used.[3] The choice of therapy is also influenced by the patient's other medical conditions, after careful evaluation of the risk-benefit ratio. Fortunately, our patient was promptly referred to a rheumatologist. Following a thorough diagnostic evaluation, systemic vasculopathy was identified as a component of both Sjögren's syndrome and Kawasaki disease. Subsequently, she commenced daily hydroxychloroquine treatment and exhibited a positive response, as evidenced by the improvement in the sizes of her femoral and iliac vessels.

Coronary artery bypass grafting (CABG) has been shown effective in revascularizing coronary lesions in both pediatric and adult KD patients, with favorable long-term outcomes and 10-year survival rates exceeding 90%.[21] Additionally, it has been associated with better arterial patency rates and lower mortality during follow-up.[21] Although no randomized trials of CABG versus PCI have been conducted in KD patients, the decision-making has largely been based on data and experience from typical atherosclerotic coronary artery disease (CAD) patients. Observational retrospective studies in the bare-metal stent era found no major differences in deaths from any cause between CABG and PCI; however, an increased need for repeat revascularization procedures was noted in the PCI group.[22] Currently, American

Heart Association consensus statements on revascularization in Kawasaki disease are based on observational data and favor CABG in older children and adults with multivessel involvement or with chronic total occlusions, utilizing bilateral internal mammary artery grafts where feasible.[11] In severe cases where interventional procedures or CABG are not feasible, cardiac transplantation may be considered, especially in patients with irreversible myocardial dysfunction and complex coronary artery lesions.[11,23] Although a last-resort option due to its complexity and associated risks, transplantation can offer a viable solution for patients facing severe complications.

In conclusion, this case report emphasized the need for multi-disciplinary management of KD with systemic vasculopathy and the importance of patience, intervention skills, and an advanced treatment toolbox to address the complex coronary lesions and systemic vascular disease in such patients.

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