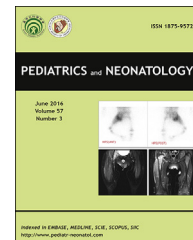


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BRIEF COMMUNICATION

Rotational Atherectomy and Stent Implantation in an 11-Year-Old Boy with a History of Kawasaki Disease



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1. Introduction

Percutaneous transluminal coronary rotational atherectomy (PTCRA) is a coronary intervention in adults with coronary arterial stenosis caused by heavily calcified atheroma.¹ Kawasaki disease (KD) is one of the most common forms of pediatric systemic vasculitis.² Despite receiving intravenous immunoglobulin therapy, coronary arterial lesions occur in 5–20% of patients with acute-stage KD.^{2,3} Coronary lesions of KD may persist and even progress to stenosis or obstruction.² One study reported that aneurysms larger than 8 mm (so-called “giant aneurysms”) did not regress during a 30-year follow-up period.³ The acute myocardial infarction/death-free survival rate was only 0.76% and 0.69% at 10 years and 20 years after KD onset, respectively. A previous report has suggested that PTCRA

could be effective for calcified localized stenosis in the chronic stage of KD.² However, to the best of our knowledge, no similar study has been conducted in Taiwan. Herein, we report the results of PTCRA for calcified lesions in an 11-year-old Taiwanese boy with KD.

2. Case Report

An 11-year-old boy (31 kg) experienced two separate episodes of KD at the ages of 8 months and 11 months. Cardiac sonography at the age of 11 months revealed giant aneurysms of the left main coronary artery (LMCA; 7.1 mm) and the right coronary artery (RCA; 7.3 mm). Cardiac catheterization at the age of 8 years revealed aneurysm of the LMCA, small aneurysm (3.4 mm) of the left circumflex coronary artery (LCX) with 40% of stenosis (Figure S1A), and a recanalized RCA with bridging collaterals (Figure S1B). The stress cardiac scintigram (Tl²⁰¹) showed a mild reversible perfusion defect at the mid to basal inferior wall. The patient was regularly taking the following medication: a low dose of aspirin (3 mg/kg/d), warfarin (2.5 mg once a day), and diltiazem (15 mg twice a day).

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In May 2013, the boy, then aged 11 years, experienced several angina attacks and cold sweating at school and late at night. Although emergency laboratory data were within the normal range (no ST changes on the electrocardiogram and no elevation of cardiac enzymes), selective coronary angiography was planned to define any potential progression of coronary lesions. Femoral vascular access was established, and heparin (50 units/kg) was administered. Additional boluses (25 units/kg) of heparin were administered during the procedure to maintain an activated clotting time of more than 200 seconds. Selective RCA angiography revealed similar findings to those of the examinations 3 years previously. Left coronary cineangiogram demonstrated: (1) 75% stenosis at the proximal site of the LCX (white arrowhead in [Figure 1A](#)); (2) a giant aneurysm of the LMCA with wall calcification (white arrow in [Figure 1A](#)); and (3) flow through small collaterals from the LCX to the distal portion of the occluded RCA ([Figure S2](#)).

The left coronary ostium was engaged with a 6-Fr JL3.5 guide catheter for subsequent percutaneous transluminal coronary angioplasty (PTCA) and possible rotational atherectomy. A Rinato wire (Asahi Intecc, Aichi, Japan) was used to cross the stenotic lesion in the LCX. Angioplasty was initially performed with a 2.5 mm × 12 mm Trek balloon (Abbott Vascular, Santa Clara, CA, USA), a 2.5 mm × 15 mm Trek balloon, and then a 2.5 mm × 12 mm NC Sprinter balloon (Medtronic, Minneapolis, MN, USA) up to 16 atm. However, we failed to dilate the obstruction by using a balloon angioplasty ([Figure 1B](#)), and dissection was noted with an easily collapsed intima flap and compromised flow (black arrow in [Figure 1C](#)). Subsequently, we performed rotational atherectomy with a burr size of 1.25 mm ([Figure 1D](#)). The burr was advanced into and successfully passed through the lesion at 169,000 rotations per minute for 15 seconds. After rotational atherectomy, a 2.0 mm × 8 mm Voyager NC balloon (Abbott) and a 2.5 mm × 12 mm NC Quantum Maverick balloon (Boston Scientific, Marlborough, MA, USA) were used to perform PTCA. The waist finally disappeared. Thereafter, we deployed a 2.5 mm × 14 mm Biomatrix stent (Biosensors, Singapore, Singapore; [Figure 1E](#)) with the balloon inflated to a pressure of 6 atm, and then performed a poststenting dilatation with a 2.5 mm NC Quantum Maverick balloon (Boston Scientific). Excellent antegrade flow and no residual narrowing were noted after stent deployment ([Figure 1F](#)). Follow-up intravascular ultrasound images revealed acceptable opposition of the stent to the wall of the LCX. After the procedure, the child was transferred to an intensive care unit for monitoring and supportive care. T-wave inversion was noted in the V5 and V6 leads but recovered rapidly 1 day later. An elevated creatinine kinase level (786 mg/dL) was detected immediately after the procedure but normalized 2 days later. Clopidogrel at 37.5 mg and warfarin at 2.5 mg were administered once daily as antiplatelet and anticoagulation therapies. Computed tomography performed 1 year after the procedure revealed improved flow in the distal LCX.

3. Discussion

We successfully performed rotational atherectomy followed by stent implantation in an 11-year-old boy with

three-vessel coronary disease and calcified coronary stenosis caused by KD. To the best of our knowledge, this is the first pediatric patient with KD to undergo PTCRA and stent placement in Taiwan. This treatment approach is an alternative to coronary artery bypass grafting in children with KD.

A pathological study⁴ has shown that coronary arterial stenosis after KD is caused by marked intimal thickening and multiple calcifications at the site of aneurysms and stenosis. Such thickening and calcification reduces the compliance of the arterial wall and compromises the success of PTCA in patients with KD.⁵ In this patient, we failed to advance the follow-up intravascular ultrasound catheter to the distal lumen of the LCX stenosis before PTCRA and used a 2.5 mm noncompliant Sprinter balloon (Medtronic) to conduct PTCA with a maximal balloon pressure of 16 atm. We not only failed to dilate the stenosis but also created an intima flap and serious dissection, which is a major cause of new traumatic coronary aneurysms after PTCA in patients with KD.^{2,5} The findings of the current case are supported by the guidelines of the Japanese Circulation Society,² which recommend that the balloon pressure should be less than 10 atm, and that balloon dilatation is more effective for treating stenotic lesions developed within 6 years after KD onset. PTCRA is preferred when calcification exceeds 50% of the lumen or when severe stenosis fails to respond to balloon dilatation.

The timing of the intervention is crucial. A coronary cineangiogram of this patient 3 years earlier (at the age of 8 years; [Figure S2](#)) had already revealed an aneurysm of the LMCA, a recanalized RCA caused by bridging collaterals, the stenotic LCX (40%), and small collateral from the LCX to distal RCA. A concurrent thallium-201 perfusion scan also showed the perfusion defect at the mid to basal inferior wall. Some researchers⁴ have claimed that the catheter intervention should be considered for angiographically significant coronary stenotic (>50%) lesions in patients with KD who have no symptoms, because half of the ischemic events occurred in the patients with KD who did not have any previous ischemic findings. However, complications of PTCA⁶ and PTCRA, such as restenosis, total occlusion, and the development of new aneurysms, have also been reported in patients with KD. Currently, the Japanese Circulation Society² strongly recommends catheter-based therapy whenever coronary angiographic studies (computed tomography, magnetic resonance imaging, or catheterization) show significant stenosis (≥75% of luminal diameter) and stress tests reveal ischemic symptoms or findings. When the patient was aged 8 years, we increased the dosage of anticoagulants and administered calcium channel blockers rather than performing a coronary intervention. More frequent follow-up (e.g., every 1 year or 2 years), computed tomography, or magnetic resonance imaging for patients with KD and a known history of coronary stenosis, similar to our case, may promote the earlier detection of coronary stenosis progression and even increase the probability of successful PTCA in such patients.^{2,6}

Coronary artery bypass grafting (CABG) is one of the main treatment options for the revascularization of calcified coronary arterial stenosis after KD. Long-term CABG

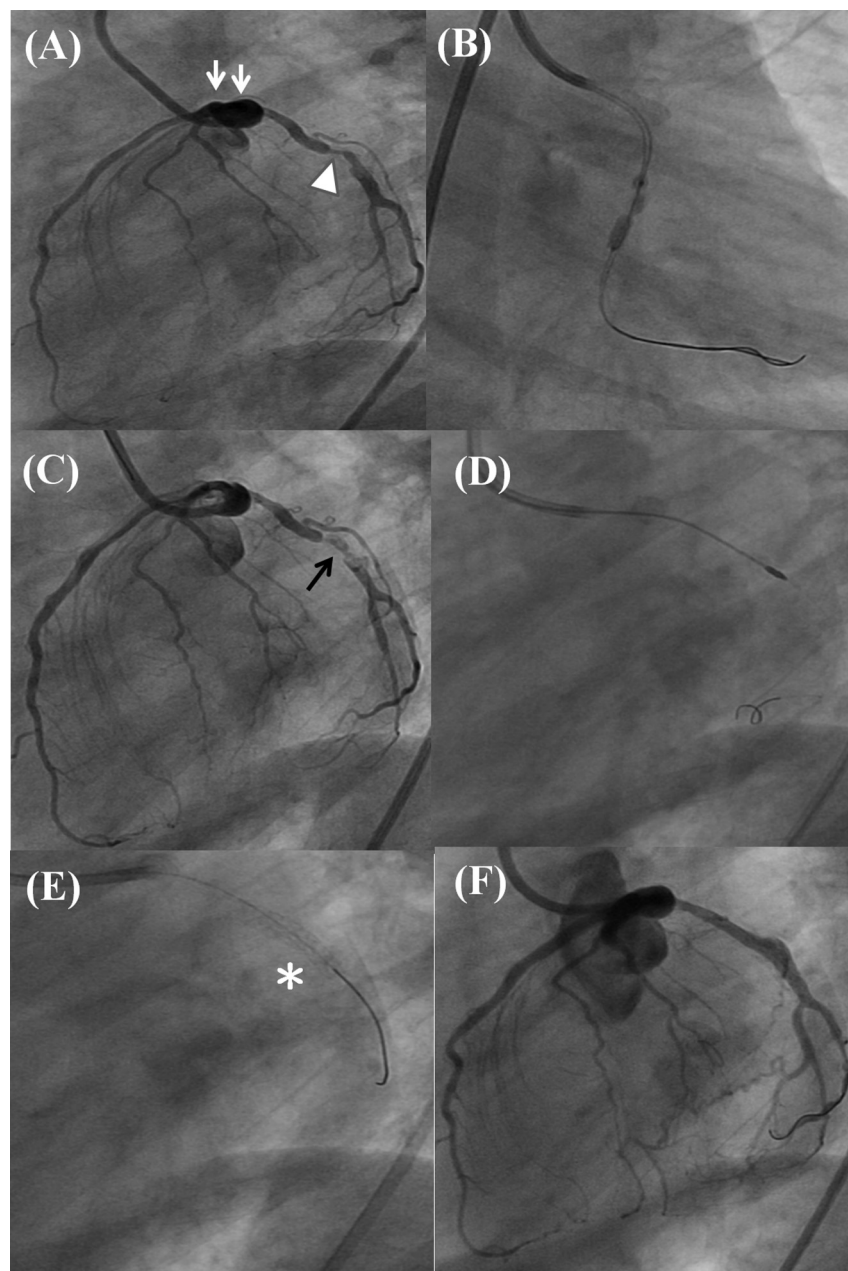


Figure 1 Angiogram of the left main coronary artery at the age of 11 years; a giant aneurysm of the left main coronary artery is noted (white arrow) (A, C–F: Left anterior oblique 60° + Caudal 20°; B: Right anterior oblique 30° + Caudal 20°). (A) Seventy-five percent stenosis of the left circumflex coronary artery (white arrowhead); (B) limited effect of balloon angioplasty; (C) intima flap and dissection (black arrowhead); (D) rotational atherectomy with a burr size of 1.25 mm; (E) stent implantation after rotational ablation; and (F) cineangiogram showing the restored left circumflex coronary artery flow and no stenosis.

results in pediatric patients with KD are favorable, with one study reporting a 20-year survival rate of 95%.⁷ However, in the same study, the cardiac event-free rates declined from 85% (10 years) to 68% (25 years).⁷ Furthermore, the long-term rate of graft patency (internal thoracic artery) was relatively higher in patients with KD who were older than 10 years (93%), compared with the rate in those younger than 10 years (86%). According to the aforementioned findings, we decided to adopt the staged approach (PTCRA followed by stent implantation) to defer a possible CABG procedure

until the patient reached young adulthood. In conclusion, we demonstrated that PTCRA followed by stent implantation is feasible in a child with KD who has angina associated with severe calcified coronary stenosis.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.pedneo.2015.07.005>.