Cardiology in the Young

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Long-term results of percutaneous transluminal coronary balloon angioplasty in Kawasaki disease

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Brief Report

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Abstract

We report the long-term results of percutaneous transluminal coronary balloon angioplasty for early appearing localised stenosis of the left anterior descending artery caused by Kawasaki disease in two 2-year-olds. One patient had 50% restenosis and newly appearing aneurysm at 21-year-old, and the other had no restenosis at 16-year-old. They had no symptoms or cardiac events for 20 years. It is useful in the early localised stenosis without aneurysm.

Percutaneous transluminal coronary balloon angioplasty is ineffective for localised stenosis with calcification in the late period after Kawasaki disease.[1] However, some successful results of percutaneous transluminal coronary balloon angioplasty in children were reported in the 1990s.[2–5] We report the long-term results of percutaneous transluminal coronary balloon angioplasty and the morphological changes of large aneurysms over 14 years in two grown-up patients.

Patient 1

A 2.7-year-old girl, with a body weight of 12.8 kg, had Kawasaki disease at the age of 4 months (Table 1). Although intravenous immunoglobulin was administered, she developed bilateral giant aneurysms. The maximum diameters of the right coronary artery and left anterior descending artery in selective coronary angiogram at the age of 7 months were 8.4 mm (Z score 13.8) and 9.2 mm (Z score 20.0), respectively (Fig 1).[6] Coronary angiograms at the age of 2 years revealed severe localised stenosis in segment 6 of the left anterior descending artery. An early defect suggestive of ischemia in the anteroseptal wall of the left ventricle was detected on dipyridamole-loaded electron beam CT. No calcification of the coronary arteries was detected on CT. She underwent percutaneous transluminal coronary balloon angioplasty for severe localised stenosis under general anesthesia. The diameter of the balloon catheter was 2.5 mm, and the balloon was inflated twice, for 35 and 30 seconds, to a pressure of 8 atm through the 6-F guiding catheter. The localised stenosis improved from 80% to 21% (Fig 1). Intravascular ultrasound showed severe intima-medial thickening in the localised stenosis before percutaneous transluminal coronary balloon angioplasty and the intima-medial layer was a low echoic area.[5] The intima-medial thickness, internal lumen area, and vascular area were 1.80 mm, 1.08 mm^2, and 14.8 mm^2, respectively. After percutaneous transluminal coronary balloon angioplasty, the intima-medial thickness, internal lumen area, and vascular area were 1.09 mm, 2.28 mm^2, and 14.5 mm^2, respectively. The thickened intima-medial layer was compressed and the internal lumen area was increased without a change in the coronary artery area. Ischemia was improved in an electron beam CT. Aspirin was administered as antiplatelet therapy, and warfarin was stopped. Nifedipin was added for 1 year. Repeat coronary angiogram 3 years later showed no significant restenosis of the left anterior descending artery (Fig 1). Coronary angiogram at the age of 21 years revealed 50% restenosis and new aneurysm. Although the diameter of the target vessel did not change, the diameter of the proximal site was increased. The instantaneous wave-free ratio was 0.88. There was no ischemia. In the CT finding, a small calcification at the site of the new aneurysm in the left anterior descending artery was detected (Fig 1). She has had no cardiac events for 20 years after percutaneous transluminal coronary balloon angioplasty.

Patient 2

The second patient, a 2.2-year-old boy, with a body weight of 11.5 kg, had Kawasaki disease at the age of 3 months. Despite intravenous immunoglobulin, he developed bilateral large aneurysms. The maximum diameters of the right coronary artery and left anterior descending artery in coronary angiogram at the age of 5 months were 7.7 mm (Z-score 12.3)

Table 1. Patient characteristics

	Patient 1	Patient 2
Gender	Female	Male
Age at the onset of KD	4 months	3 months
Initial CAG	7 months	5 months
Weight (kg)	7.3	7.0
Height (cm)	68.8	65
Maximum diameter (mm)		
RCA (Z score)	9.2 (19.95)	7.7 (12.28)
LAD (Z score)	8.4 (13.75)	4.8 (8.70)
LCA (Z score)	5.8 (13.75)	3.5 (5.53)
Medication	Aspirin, Coumadin	Aspirin, Ticlopidine
Age at PCBA	2 years 7 months	2 years 2 months
Weight (kg)	12.8	11.5
Diameter of stenosis		
Before (%)	0.7 mm (80%)	0.6 mm (89%)
After (%)	1.5 mm (21%)	1.9 mm (0%)
Diameter of balloon (mm)	2.5	2.0
Balloon pressure (atm)	8	6, 8
Duration of inflation (s)	35, 30	30, 30
Medication after PCBA	Aspirin, Nifedipin	Aspirin, Sarpogrelate
Age at follow-up CAG	21 years	16 years
LAD	50% localized stenosis	No stenosis
RCA	50% localized stenosis	Segmental stenosis
Medication	Aspirin	Aspirin

CAG = coronary angiogram; KD = Kawasaki disease; LAD = left anterior descending artery; LCA = left coronary artery; PCBA = percutaneous transluminal coronary balloon angioplasty; RCA = right coronary artery.

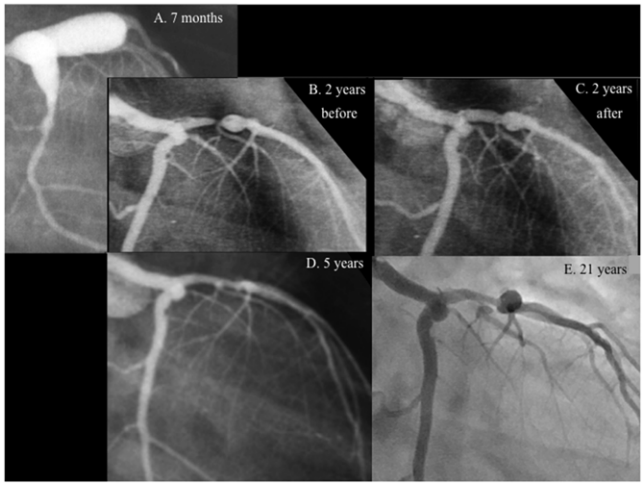

Figure 1. Follow-up coronary angiograms of the LAD in patient 1.
A, initial CAG (7 months old); B and C, before and after PCBA (2 years old); D, 5 years old; and E, 21 years old.

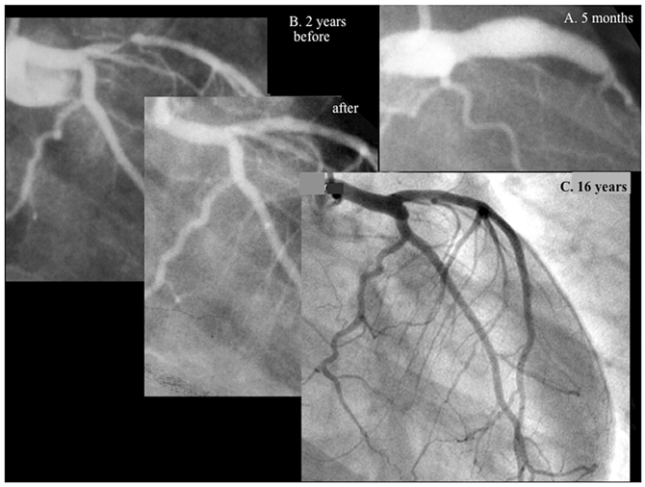

Figure 2. Follow-up coronary angiograms of the LAD in patient 2.
A, initial CAG (5 months); B, before and after PCBA (2 years old); and C, 16 years old. Collateral arteries were found from the left circumflex artery to the posterior decending artery.

and 4.8 mm (Z-score 8.7), respectively (Fig 2). Coronary angiogram at the age of 2 years revealed severe localised stenosis at segment 6 of the left anterior descending artery. ST-T depression in leads V_2–V_5 was detected in a dipyridamole-loaded electrocardiogram. An early defect in an electron beam CT was detected in the anteroseptal wall of the left ventricle, and calcification of the coronary arteries was not detected. He underwent percutaneous transluminal coronary balloon angioplasty for localised stenosis through a 5-F sheath under general anesthesia. The diameter of the balloon catheter was 2.0 mm, and the balloon was inflated twice for 30 seconds to a pressure of 6 atm and 8 atm, respectively. The localised stenosis improved from 89% to 0% (Fig 2). Ischemia was improved in an electron beam CT, electrocardiogram, and myocardial imaging after percutaneous transluminal coronary balloon angioplasty. Aspirin was administered as antiplatelet therapy. Coronary angiogram at the age of 16 years showed no evidence of restenosis of the left anterior descending artery. There was no calcification of the left anterior descending artery in the CT angiography. In regard to the right coronary artery, 50% localised stenosis was detected at the age of 2 years. Three years later, the localised stenosis had progressed to 90% with calcification. Percutaneous transluminal coronary rotational ablation was performed in the patient when his body weight was 19 kg.[7] One year later, coronary angiogram revealed asymptomatic complete occlusion of the right coronary artery. Coronary angiogram at the age of 16 years revealed segmental stenosis which implied coronary recanalisation after thrombotic coronary artery occlusion.

Discussion

In coronary artery stenosis in Kawasaki disease, intimal thickening develops due to fibrocellular proliferation during the convalescent stage after destruction of the vessel wall by acute inflammation. The time of appearance of localised stenosis varies, ranging from several months to more than 10 years after the onset of Kawasaki disease.[8] Coronary artery calcification increases with ageing after Kawasaki disease.[9] When calcification of the affected coronary arteries was not detected either by fluoroscopy or CT, a low echoic intima-medial appearance by intravascular ultrasound indicates a rich extracellar matrix making percutaneous transluminal

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coronary balloon angioplasty possible for severe localised stenosis. Percutaneous transluminal coronary balloon angioplasty in Kawasaki disease would be more effective in shorter and earlier localised stenosis. Previous reports have suggested asymptomatic occlusion 1 year after percutaneous transluminal coronary balloon angioplasty in patients with localised stenosis and large aneurysm. Coronary occulsion may be caused by thrombotic occlusion rather than intimal thickening of coronary artery wall thickness after percutaneous transluminal coronary balloon angioplasty. It may be likely to occlude after percutaneous transluminal coronary balloon angioplasty in patients with localised stenosis and aneurysm. In such patients, coronary artery bypass grafting would be recommended.

One patient had 50% restenosis and new aneurysm (patient 1) and the other had no restenosis (patient 2) in the late period. Although there was a small calcification in the left anterior descending artery in patient 1, there was no calcification in the left anterior descending artery in patient 2. The maximum diameter of the left anterior descending artery in the initial coronary angiogram in patient 1 was 8.4 mm (Z-score 13.8), and that in patient 2 was 4.8 mm (Z-score 8.7). It is speculated that one of the causes of the difference in the morphological changes after percutaneous transluminal coronary balloon angioplasty may depend on the degree of involvement of coronary artery wall in the acute phase of Kawasaki disease.[6] A newly appearing aneurysm in the late period is often formed at the site of the stenosis where the aneurysm had regressed.[10] They can also be found in a bifurcation of branching site. It is speculated that the haemodynamic changes due to the stenosis in the late period related to the causes of newly appearing aneurysms. Newly appearing aneurysms may be recognised as "re-appearing aneurysms."

Conclusion

Percutaneous transluminal coronary balloon angioplasty in small children for early appearing localised stenosis without aneurysm due to intimal thickening 1–2 years after the onset of Kawasaki disease can provide a long-term patency for 20 years. Percutaneous transluminal coronary balloon angioplasty may be a useful procedure in such patients, although its indication is limited.

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Conflicts of interest. The authors state that they have no conflict of interest.

Ethical Standards. All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional committee with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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