A 25-year-old woman was admitted to our hospital because of cardiac arrest. She was a non-smoker, had no hypertension or diabetes mellitus, but did have dyslipidemia. She did not take any medicines before admission. She had a fever lasting approximately 1 week, and she had developed a rash at approximately 2 years old, but she was never diagnosed as having Kawasaki disease (KD). She complained of chest pain and had cardiac arrest. Although circulatory function returned spontaneously, disturbance of consciousness continued (Japan coma scale score, 20). Electrocardiogram showed ST elevation in II, III, aVF, and laboratory data indicated elevated CK at 255 IU/L (CK-MB, 36 IU/L). In addition, low-density lipoprotein cholesterol (LDL-C) was 61 mg/dL, and high-
density lipoprotein cholesterol (HDL-C) was 30 mg/dL at 2 days after treatment with pitavasatin Ca 2 mg.

We assessed her for hypoxic encephalopathy because of acute myocardial infarction. Cerebral hypothermia therapy was started, followed by emergency coronary angiography (CAG). Coronary aneurysm was observed in the midportion of the left anterior descending artery (Figure 1A). The right coronary artery (RCA) had no collateral vessels. Additionally, TIMI 0 flow was detected in the proximal portion of the RCA (Figure 1B), just distal to the site of the coronary aneurysm. Aspiration was performed several times (Figure 1C). Finally, we stented the proximal portion of the RCA with a bare metal stent (Integrity™, Medtronic; Figure 1C').

The RCA had mural red thrombi and fragile yellow color (Figure 1D). Coronary angioscopy (CAS) was also performed to assess the intra-coronary condition, because we suspected that the patient had KD (Figure 2A). In the proximal aneurysm, many red thrombi were observed adhering to the aneurysmal wall, which had an intense yellow color (Figure 2a,b). An aneurysm in the midportion of the RCA had mural red thrombi (Figure 2e) and fragile white thrombi (Figure 2f). Additionally, atherosclerotic yellow plaque was present in the coronary artery aneurysm and coronary artery, which appeared normal (Figure 2c).

Fortunately, the patient was discharged with no obvious higher brain dysfunction. After percutaneous coronary intervention, dual anti-platelet therapy (aspirin, 100 mg/day; clopidogrel, 75 mg/day), warfarin, and statin (pitavastatin 2 mg) were used for 1 month. Then aspirin, warfarin, and statin were continued. International normalized ratio of prothrombin time was controlled between 2.0 and 3.0 using warfarin. Subsequently, the patient underwent coronary computed tomography, which showed no calcification inside the coronary aneurysm, but calcification was found at a seemingly normal site on CAG (Figure 1E,F).

Six months later, the patient underwent follow-up CAG under aspirin, warfarin, and statin. At follow-up, LDL-C was 69 mg/dL, and HDL-C was 41 mg/dL. According to follow-up CAG, in-stent restenosis was not detected. On CAS to assess the RCA after intensive medical intervention, the yellow plaque had regressed and turned white, even in the coronary aneurysm (Figure 2a'–f').

This is the first report to describe the assessment of a coronary aneurysm caused by KD using CAS. The present young woman with myocardial infarction had no coronary risk factors, but a history of KD was suspected, and in the present case it was thought that acute coronary syndrome (ACS) was due to atherosclerotic change by KD.

KD is a systemic vasculitis of unknown cause that occurs primarily in children <6 years old. Cardiovascular complications in the acute phase occur in 9.3% of patients, and 3.0% experience cardiovascular sequelae. Cardiovascular sequelae include coronary dilatation (1.90%) and aneurysms (0.78%).

The long-term survival in KD complicated by giant coronary aneurysms is moderately good with multiple catheter and surgical interventions. There are cases, however, in which KD was not diagnosed in advance and treatment with i.v. Ig had not been provided. Coronary artery lesions occur in 15–20% of untreated patients. The present patient was thought to have developed coronary aneurysms because of a history of KD.

In the current patient, the continuation of aspirin and a statin was thought to stabilize the atherosclerotic plaque. And, in fact, the coronary plaque changed from yellow to white. Statin use stabilizes atherosclerosis, and the reduction of LDL-C by ≥50% or maintenance of LDL-C to ≤70 mg/dL through lipid-lowering therapy is sufficient to prevent atherosclerosis. The inflammation of vessels due to KD is a serious lifetime problem, and 18fluorodeoxyglucose positron emission tomography (FDG-PET) has been reported to be efficient for detecting vascular inflammation in KD.

Furthermore, statin therapy has been reported to reduce
persistent coronary arterial inflammation due to KD on serial $^{18}$FDG-PET. The present case suggests that even if atherosclerosis has stabilized in patients with KD, careful follow-up should be continued to prevent ACS.

**Funding**

None.

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