Early-Period Coronary Aneurysm Formation After Sirolimus-Eluting Stent Implantation

A 69-year-old male patient presented to the emergency department with chest pain. The high sensitive-troponin T level was found to be 156.7 pg/mL (0-19 pg/mL) in the patient, with no significant ischemic change in his electrocardiography. It was learned that the patient with a history of hypertension, dyslipidemia, and diabetes mellitus had undergone drug eluting stents (DES) (Star Supraflex Stent, Sahajanand Medical Technologies Ltd., Sahajanand Estate, Wakharwadi, Near Dabholi, India) implantation to the left anterior descending (LAD) artery 7 days ago in another center due to non-ST elevation myocardial infarction (Figures 1A, 1B). After stent implantation, it was observed that the D1 branch was occluded, but a medical decision was made because of the small calibration.

The stent extending from the proximal LAD to the D2 branch segment was open in his coronary angiography. The fusiform coronary aneurysmatic formation was observed in the proximal part of the stent. Also, an aneurysmatic sac was observed in the distal part of the stent. The D2 ostial was mildly thrombotic, but TIMI 3 flow was present (Figures 2A, 2B). Anticoagulant and IV antiplatelet therapy was administered, and a decision was made for medical follow-up. Coronary computed tomography angiography was applied to the asymptomatic patient 2 weeks after discharge. It was observed that the size of the aneurysm continued without increasing. The proximal aneurysm diameter was 8.5 × 6 mm and the distal aneurysm diameter was 4 × 3.3 mm (Figures 3A, 3B, Video 1).

Acquired coronary artery aneurysm (CAA) may occur because of atherosclerosis, Kawasaki disease, Takayasu arteritis, other connective tissue diseases, infections, trauma, percutaneous coronary intervention (PCI), and DES implantation.1-4 Mechanical vessel wall injuries during PCI (balloons, stents, and atherectomy devices) and eosinophil-rich vascular inflammatory reactions against polymers, nickel, cobalt, and stent-released drugs have been thought to be associated with the development of CAA after PCI.2

Although the polymers used for drug delivery in new-generation DES are highly biocompatible, they can rarely trigger a severe inflammatory reaction consisting of eosinophils and lymphocytes, including all 3 arterial layers, leading to aneurysm formation and significantly increasing the risk of stent thrombosis.5
However, the case of aneurysm development at such an early stage is infrequent. The development of CAA was demonstrated 1 week after sirolimus-eluting stent implantation in our case.