A 72-year-old female patient was referred from a local hospital because of a sudden cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed for 7 min, and the patient was resuscitated. After being transferred, she was monitored at the intensive care unit and evaluated to determine the cause of the sudden cardiac arrest. Coronary angiography revealed a significant stenosis extending from the distal left main to the proximal left anterior descending (LAD) coronary artery. The right coronary artery (RCA) showed a significant diffuse stenosis. The most severe discrete stenosis was present at the distal RCA to the posterior lateral (PL) branch lesion. We performed PCI with stenting at the short segment of the distal RCA to the PL branch. Then, we conducted an IVUS-guided PCI with stenting from the distal left main to the proximal LAD crossing over to the left circumflex (LCx) coronary artery (Fig. 1B). The IVUS image showed an eccentric echodense plaque with severe luminal narrowing at the proximal LAD, and no severe stenosis or plaque rupture was seen at the proximal LCx coronary artery. After the procedure, the patient’s blood pressure gradually decreased, and she suddenly developed an atrioventricular block. We inserted a temporary pacemaker and started vasopressor therapy. Another coronary angiography showed total occlusion of the proximal RCA, causing severe spasm. We injected nicorandil, which partially resolved the spasm, and the patient’s blood pressure was elevated, and her heart rhythm was converted to sinus rhythm. IVUS of the RCA revealed diffuse intima and media thickening with a large amount of atheromatous plaque at the mid-RCA level. In the spasm portion of the proximal RCA, there was a large area of attenuated plaque and prominent intima thickening with rupture. Therefore, we performed PCI with stenting at the proximal RCA plaque rupture lesion. After stenting, the RCA flow was well maintained, and the patient was stabilized. We observed the patient for several minutes; however, her blood pressure suddenly decreased, and she again developed a pacing rhythm. We performed an RCA coronary angiography again, and the deployed stent remained patent and the flow was maintained well. We performed a left coronary angiography, which revealed total occlusion of the LCx coronary artery. We injected nicorandil and the flow was fully resolved abruptly; however, the patient again developed a spasm. Her blood pressure was continuously low, and she developed ventricular fibrillation. We performed CPR and applied PCPS, and the patient was stabilized thereafter. We conducted left coronary angiography to confirm the patency of the LCx coronary artery. However, coronary spasm again developed at the mid-RCA (Fig. 5A) and it could not be resolved despite multiple injections of nicorandil. We deployed a stent at the mid-RCA to mechanically stop the spasm. The flow was recovered after stenting. After 7 days, the patient could be weaned from the PCPS and she was discharged 1 month later. Three months later, she was admitted for a transient ischemic attack. During the hospital stay, she experienced a gastrointestinal bleeding event and heart failure aggravation. We stopped the diltiazem medication, which was the cause of reduced heart function. On the next day, she had a sudden cardiac arrest again and was resuscitated after a 5-min CPR. We intravenously injected nitrate and performed coronary angiography. All stents were patent, and no other lesion progression was found. The patient recovered and was discharged with prescriptions of clopidogrel, ramipril, statin, diltiazem, nicorandil, and isosorbide dinitrate.