

Visualization of a Rapidly Progressing Lipid-Rich Plaque Causing No-Reflow Phenomenon During Percutaneous Coronary Intervention

— Insights From Near-Infrared Spectroscopy and Histopathology —

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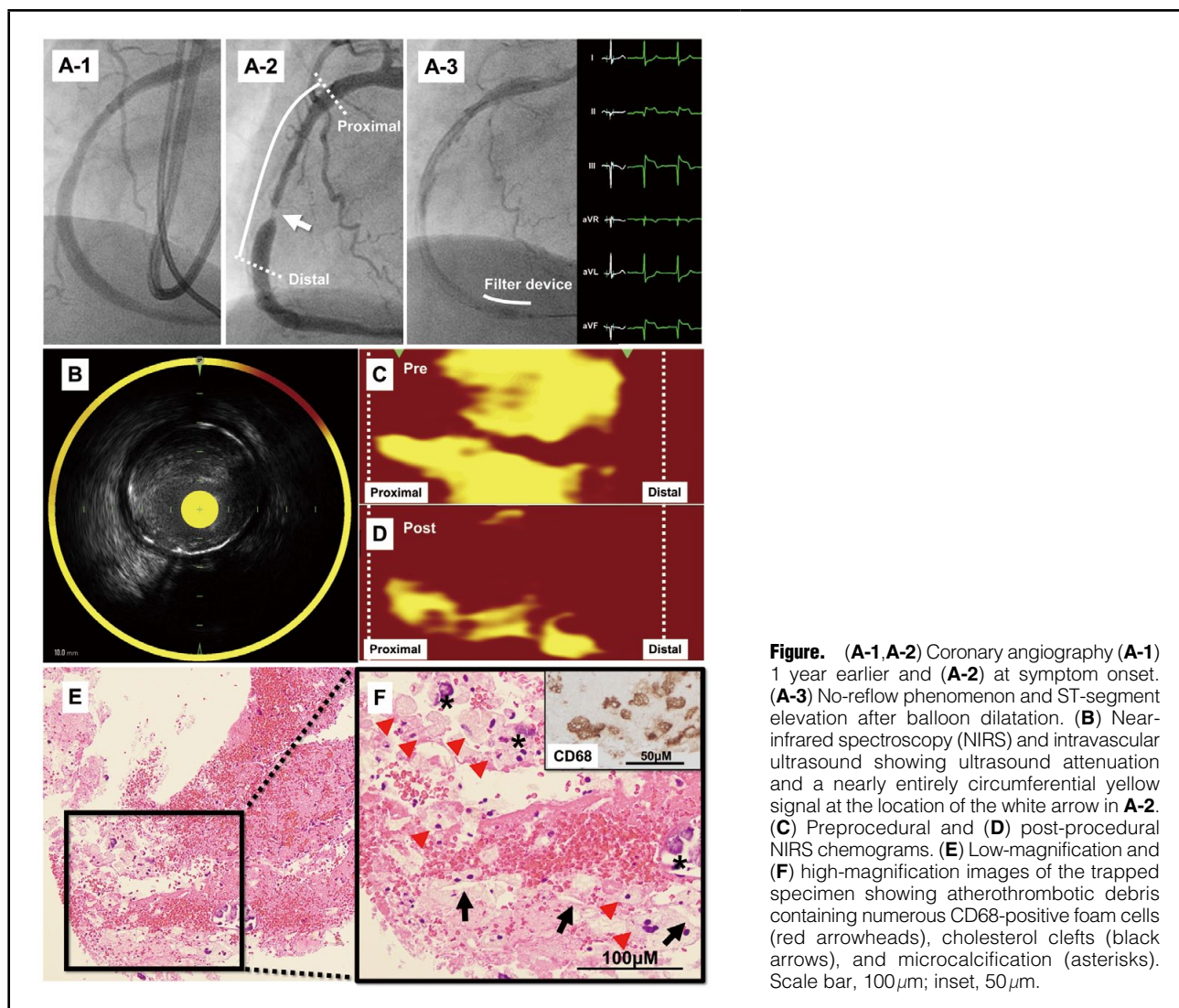


Figure. (A-1,A-2) Coronary angiography (A-1) 1 year earlier and (A-2) at symptom onset. (A-3) No-reflow phenomenon and ST-segment elevation after balloon dilatation. (B) Near-infrared spectroscopy (NIRS) and intravascular ultrasound showing ultrasound attenuation and a nearly entirely circumferential yellow signal at the location of the white arrow in A-2. (C) Preprocedural and (D) post-procedural NIRS chemograms. (E) Low-magnification and (F) high-magnification images of the trapped specimen showing atherothrombotic debris containing numerous CD68-positive foam cells (red arrowheads), cholesterol clefts (black arrows), and microcalcification (asterisks). Scale bar, 100µm; inset, 50µm.

Received October 10, 2019; revised manuscript received November 21, 2019; accepted December 5, 2019; J-STAGE Advance Publication released online December 17, 2019 Time for primary review: 28 days
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A 72-year-old man with a history of myocardial infarction was hospitalized due to angina pectoris. Coronary angiography identified progressive severe stenosis in the middle segment of the right coronary artery where intermediate stenosis had been observed 1 year earlier (**Figure A-1,A-2; Supplementary Movie 1**). Near-infrared spectroscopy (NIRS) and intravascular ultrasound showed ultrasound attenuation (**Figure B**) with a high maximum 4-mm lipid core burden index (max-LCBI) of 963 (**Figure C**). A filter-based distal protection device (DPD) was used before balloon dilatation due to high LCBI. Following balloon dilatation of the target lesion, the patient complained of chest pain. Electrocardiography (ECG) showed ST-segment elevation in the inferior leads. Coronary angiography demonstrated no-reflow phenomenon (**Figure A-3; Supplementary Movie 2**). Next, a sirolimus-eluting stent was implanted at the lesion. Coronary flow and ECG changes completely resolved after DPD collection. NIRS demonstrated a marked reduction of yellow

signals (max-LCBI, 295; **Figure D**), suggesting that the lipid-rich plaque embolized and was trapped by the DPD. Microscopy of the trapped specimen indicated atherothrombotic debris with numerous CD68-positive foam cells, cholesterol clefts, and microcalcification (**Figure E,F**). This case clearly demonstrated a rapidly progressing lipid-rich plaque that caused no-reflow phenomenon detected on NIRS and confirmed on histopathology.

Disclosures

The authors declare no conflicts of interest.

Supplementary Files

Supplementary Movie 1. Preprocedural coronary angiography.

Supplementary Movie 2. Coronary angiography after balloon dilatation.

Please find supplementary file(s);
<http://dx.doi.org/10.1253/circrep.CR-19-0107>