An 83-year-old man was transferred to our hospital due to worsening effort angina pectoris. Electrocardiogram during chest pain at rest showed ST-segment elevation with hyperacute T waves and negative U waves in leads V2 through V4 (Figure 1A). Sublingual administration of nitroglycerin relieved him from chest pain, when electrocardiogram showed inverted T waves (Figure 1B). Biomarkers of myocardial injury were not elevated. Coronary computed tomography angiography (CTA) indicated a severe stenosis in the distal left main coronary artery (LMCA) to the proximal left anterior descending artery (LAD) (Figure 1C, yellow arrows). 18F-fluorodeoxyglucose-positron emission tomography (FDG-PET) combined with coronary CTA demonstrated the intense FDG uptake at the corresponding segment (Figure 1D, red arrows). Selective coronary angiography of the left coronary artery showed an ulcer-like severe stenosis in the distal LMCA to the proximal LAD, which we considered as a culprit lesion (Figure 1E). Next, we evaluated the coronary atherosclerotic plaque by grayscale intravascular ultrasound (IVUS) (Figures 1F to 1H), IVUS-virtual histology (Figures 1I to 1K) and optical coherence tomography (OCT) (Figures 1L to 1N). Grayscale IVUS of the culprit lesion demonstrated a substantial amount of plaque burden (Figures 1F to 1H). Fibrofatty and necrotic core components were detected within the eccentric plaque by IVUS-virtual histology (Figures 1I to 1K). OCT revealed not only a thin-cap fibroatheroma but also a dark region, suggesting a macrophage-rich region at the distal LMCA (Figure 1N, red arrow). After we evaluated the atherosclerotic plaque images, we successfully performed percutaneous coronary intervention without concomitant complication. This is the first documentation of in vivo molecular imaging of ruptured coronary atherosclerotic plaque by IVUS, OCT, and FDG-PET/CT.

KEY WORDS IVUS, macrophage, OCT, FDG-PET/CT plaque inflammation, ruptured plaque

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(A, B) Electrocardiography during chest pain and after sublingual administration of nitroglycerin. (C) Computed tomography angiography (CTA) demonstrated a severe stenosis in the distal left main coronary artery (LMCA) to the proximal left anterior descending artery (LAD) (yellow arrows). (D) 18F-Fluorodeoxyglucose-positron emission tomography (FDG-PET) combined with coronary CTA demonstrated intense FDG uptake at the corresponding segment (red arrows). Magnified views in each right side. (E) Selective coronary angiography of the left coronary artery showed an ulcer-like severe stenosis in the distal LMCA to the proximal LAD, which was a culprit lesion. (F to N) Images at the culprit lesion of grayscale intravascular ultrasound (IVUS) (F to H), IVUS-virtual histology (I to K), and optical coherence tomography (L to N). Magnified views shown on the right side. Grayscale-IVUS demonstrated a substantial amount of plaque burden (F to H). Fibro-fatty and necrotic core components were detected within the eccentric plaque by IVUS-virtual histology (I to K). Optical coherence tomography revealed not only a thin-cap fibroatheroma but also a dark region, suggesting a macrophage-rich region at the distal LMCA (N, red arrow).

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In Vivo Molecular Imaging of High-Risk Coronary Plaque