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Neoatherosclerosis with silent plaque rupture in a saphenous vein graft causing no re-flow phenomenon assessed by optical coherence tomography and histopathology

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A 79-year-old man with a history of coronary artery bypass grafting 22 years earlier was hospitalized due to stable angina. A bare metal stent was implanted in his saphenous vein graft (SVG) 15 years ago and then, a drug-eluting stent was implanted for the in-stent restenosis 8 years ago. Coronary angiography revealed a severe in-stent restenosis in the SVG anastomosed to the right coronary artery (Fig. 1A). Optical coherence tomography (OCT) showed plaque fissure, cholesterol crystals, and low-intensity area without attenuation adjacent to the lipid-rich plaque (LRP), which suggested the intraplaque hemorrhage (IPH) (Fig. 1B, a, b). OCT also revealed the disrupted intimal flap within the stent (Fig. 1B, c) and mild to moderate stenosis with LRP outside the stent. Following balloon dilatation for the target lesion, no-reflow phenomenon occurred. The coronary flow resumed by aspiration thrombectomy and nitroprusside administration. Histopathological examination of the aspirated specimens showed that they were composed of atherosclerotic plaques with fibrin, cholesterol clefts and inflammatory cells including foam cells (Fig. 1C, 1D). Previous reports have shown that IPH is one of the factors contributing to coronary plaque destabilization and plaque progression, which might cause effort angina. This case showed for the first time an in-stent neoatherosclerosis with silent plaque rupture in SVG that caused no-reflow phenomenon detected with OCT and confirmed by histopathology. Observations suggested that the use of a distal protection device and the administration of vasodilators should be strongly considered when OCT identifies the characteristics of vulnerable plaque during percutaneous coronary intervention.

Conflict of interest: None declared

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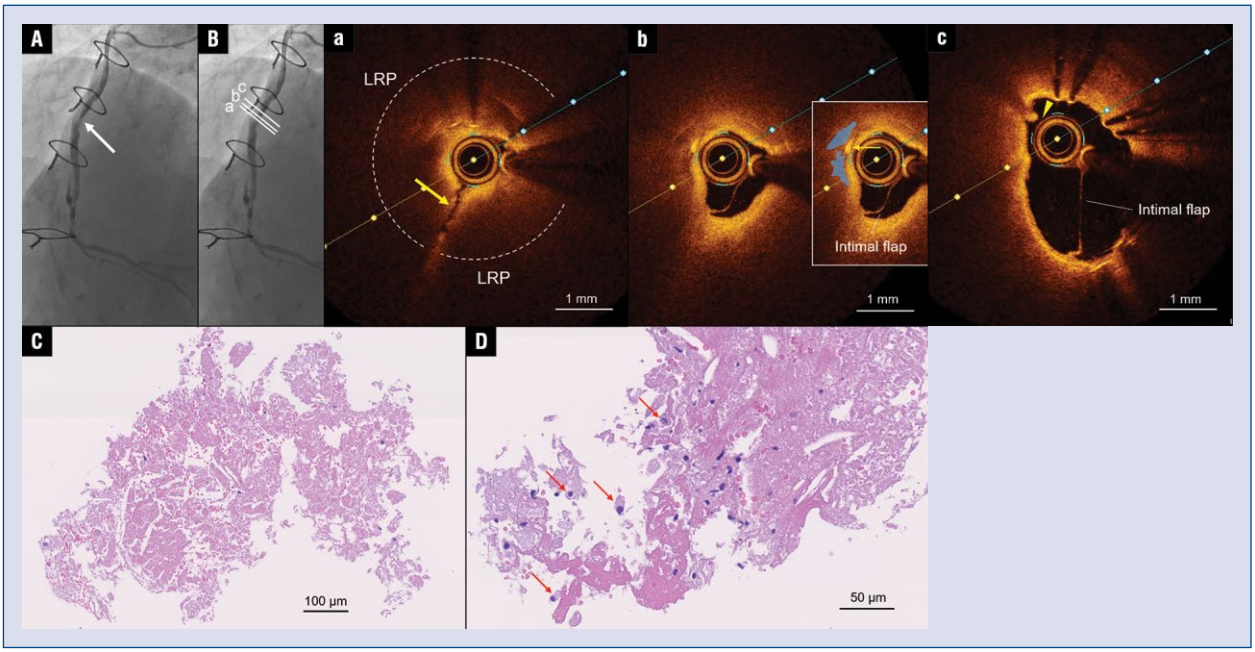

Figure 1. A. The initial coronary angiogram showing the in-stent restenosis in saphenous vein graft (white arrow). **B.** Optical coherence tomography images showing (**a**) plaque fissure (thick yellow arrow), lipid-rich plaque (LRP), (**b**) cholesterol crystals (thin yellow arrow), intraplaque hemorrhage (light blue area), and (**c**) disruption of the intimal flap (yellow arrowhead). **C, D.** Histopathology of aspirated specimens showing fibrin thrombi with cholesterol clefts and inflammatory cells including foam cells (red arrows).