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Vulnerable plaque derived from aspirated thrombi in recurrent acute coronary syndrome with familial hypercholesterolemia despite intensive lipid-lowering statin therapy

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A 35-year-old man was admitted to our hospital due to acute coronary syndrome (ACS). One year before, he was diagnosed with familial hypercholesterolemia (FH), and a stent was implanted in his left anterior descending artery and the left circumflex artery due to ACS. Despite receiving combination therapy of rosuvastatin 20 mg/day and ezetimibe 10 mg/day, his low-density lipoprotein cholesterol (LDL-C) concentration was 100 mg/dL at the second ACS. Emergency coronary angiography (CAG) revealed total occlusion of the middle right coronary artery (Fig. 1A). An intravascular ultrasound study suggested plaque rupture (Fig. 1B, arrow). Aspiration thrombectomy and stent implantation facilitated a grade 3 Thrombolysis in Myocardial Infarction flow in the final CAG. Histopathological examination of the aspirated thrombi showed a luminal thrombus (arrowhead) communicating with the underlying necrotic core with numerous cholesterol clefts (Fig. 1C-a). Inflammatory cells, including foamy and hemosiderin-laden macrophages, were also observed (Fig. 1C-b). These findings suggest that intensive lipid-lowering therapy using statin and ezetimibe was insufficient to stabilize the vulnerable plaque in a patient with FH when the targeted LDL-C level was not achieved. No further ACS has been documented after administration of proprotein convertase subtilisin/kexin type 9 inhibitor for secondary prevention over a 14-month follow-up.

Conflict of interest: None declared

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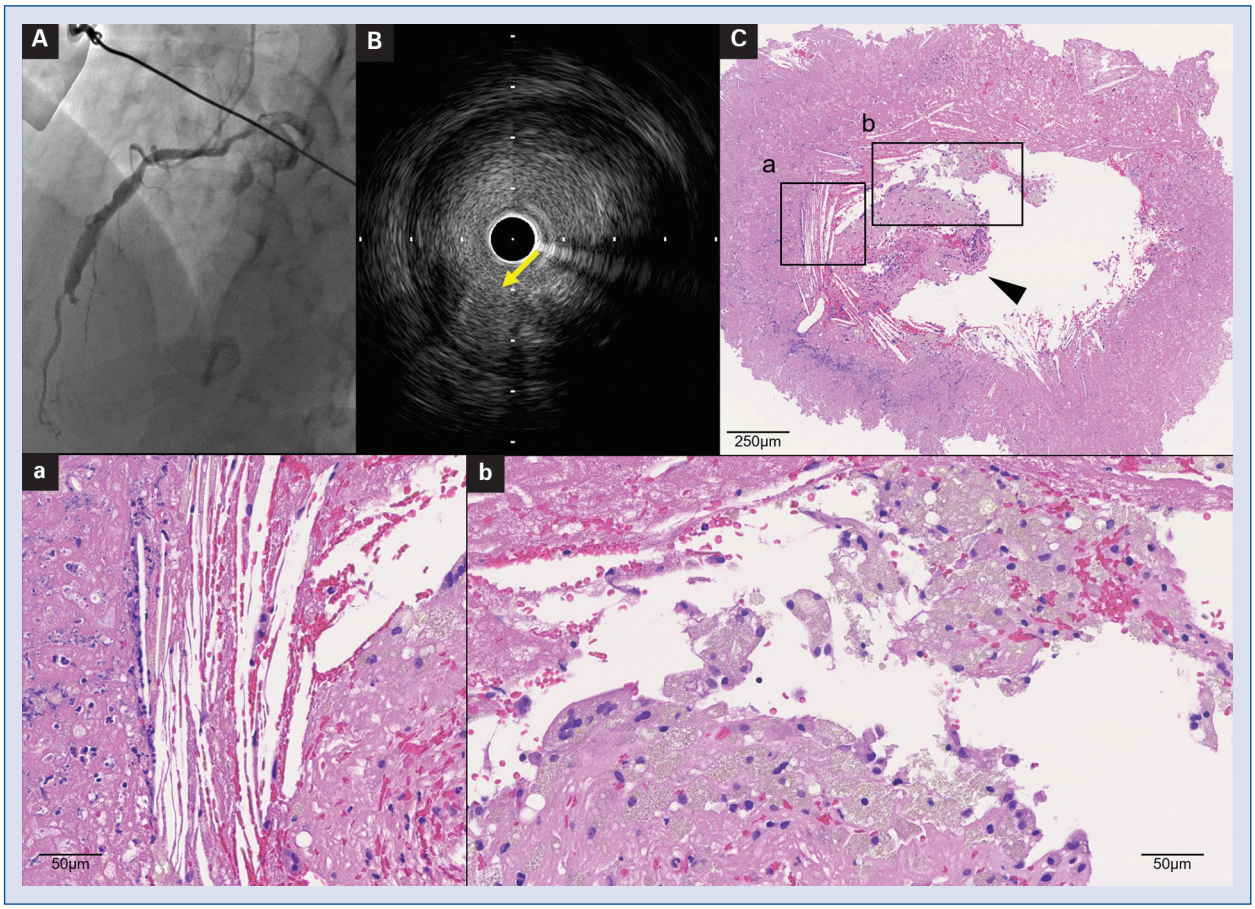

Figure 1. A. The initial coronary angiogram; **B.** The intravascular ultrasound study; **C.** Histopathology of aspirated thrombi showing (**a**) cholesterol clefts and (**b**) inflammatory cells including foamy and hemosiderin-laden macrophages.