

A Case Report Providing Insight into the Mechanism of Recurrent In-Stent Occlusion Caused by a Calcified Nodule

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A 59-year-old man on daily hemodialysis was admitted to our institution. Three years prior (X-3), he underwent percutaneous coronary intervention (PCI) with drug-eluting stent (DES) implantation for a severely calcified stenosis in the right coronary artery related to chronic coronary syndrome. Follow-up coronary angiography (CAG) one year later revealed in-stent restenosis (ISR), and repeat PCI was performed. Intravascular ultrasound (IVUS) identified a calcified nodule within the stent. Despite multiple PCIs, ISR recurred repeatedly. The patient had been on dual antiplatelet therapy (aspirin and clopidogrel) and intensive lipid-lowering therapy including a PCSK9 inhibitor.

In year X, new regional wall motion abnormalities were detected on echocardiography. CAG revealed in-stent occlusion of the right coronary artery. PCI was performed, successfully crossing the lesion with a Gladius EX guidewire. After balloon dilatation, IVUS showed two calcified nodules within the stented segment. Rotational atherectomy was employed to debulk these nodules. Although distal embolization occurred twice during the procedure, both events were effectively managed by thrombus aspiration. The procedure was completed with drug-coated balloon (DCB) inflation, achieving satisfactory vessel expansion.

Histopathological analysis of aspirated material demonstrated a fibrin network with calcification, fibrin deposits, and immature fibrous connective tissue with hemorrhage, indicating acute thrombus formation causing arterial narrowing and occlusion. Suspecting inadequate platelet inhibition by clopidogrel was a contributing factor, the antiplatelet regimen was changed from clopidogrel to prasugrel.

This case highlights how multimodality intravascular imaging combined with histopathological examination can provide valuable insights into the mechanism underlying recurrent in-stent occlusion.