A 63-year-old man with hypertension and hyperlipidemia presented to the emergency department with an infero-posterior ST-segment elevation myocardial infarction. The patient was treated with aspirin 325 mg, clopidogrel 600 mg, and intravenous heparin and was transferred for primary percutaneous coronary intervention. The coronary angiogram revealed an occluded proximal right coronary artery. Heparin was switched to bivalirudin. Aspiration thrombectomy retrieved white thrombus and established TIMI (Thrombolysis In Myocardial Infarction) grade 3 flow (Figure 1A). A 3.5×23-mm bare-metal stent was placed (Figure 1B), which was postdilated to 4.0 mm. There was no angiographic evidence of dissection or filling defect (Figure 1C) with resolution of ST-segment changes on ECG. Bivalirudin was stopped at the end of the procedure, and the patient was transferred to the cardiac intensive care unit.

Eight hours later, the patient developed recurrent chest pain with re-elevation of the ST-segment changes on ECG. An emergent coronary angiogram revealed thrombotic occlusion at the proximal edge of the stent (Figure 1D). Aspiration thrombectomy retrieved a large amount of thrombotic material, with restoration of TIMI grade 3 flow (Figure 1D, inset). Intravascular ultrasound revealed a well-expanded and well-apposed stent with no edge dissection or plaque prolapse (Figure 2). Platelet-aggregation studies revealed no evidence of resistance to clopidogrel or aspirin.

In the absence of resistance to ADP-receptor or cyclooxygenase blockade or other mechanical reasons for acute stent thrombosis, we performed near-infrared spectroscopy, a modality that detects lipid core–containing plaques. The chemogram revealed that the proximal edge of the stent ended within a large lipid-rich plaque (Figure 2). The patient was maintained on bivalirudin and eptifibatide for 12 hours after the procedure and was discharged without any subsequent events on hospital day 4, with a therapeutic regimen of aspirin 325 mg, clopidogrel 150 mg, high-dose statin, extended-release nicotinic acid, cilostazol 100 mg twice daily, and omega-3 fatty acids.

Our case highlights a possible novel clinical application of a new intravascular technology, near-infrared spectroscopy, which may implicate a disrupted lipid-rich plaque with exposed tissue factor at the edge of stents as a nidus for stent thrombosis. Near-infrared spectroscopy demonstrated the presence of residual lipid associated with acute stent thrombosis despite adequate pharmacological and mechanical management during primary percutaneous coronary intervention.
To the best of our knowledge, this is the first demonstration of acute stent thrombosis associated with residual exposed lipid from the culprit plaque. Future studies are needed to determine the precise pathogenic role of disrupted lipid-rich plaques in stent thrombosis.

**Disclosures**

None.

**References**


Residual Thrombogenic Substrate After Rupture of a Lipid-Rich Plaque: Possible Mechanism of Acute Stent Thrombosis?
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