Optical Coherence Tomography Findings in Early Stent Thrombosis by Heparin-Induced Thrombocytopenia

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Summary

A 62-year-old man with a family history of coronary artery disease and a history of smoking, diabetes and dyslipidemia was admitted to our hospital with chest pain from acute myocardial infarction. Emergent coronary angiography was performed with intervention to a mid-right coronary occlusion with drug-eluting stent implantation. Optical coherence tomography (OCT) visualized well-apposed stent struts and no remarkable tissue protrusion, stent underexpansion, malapposition, edge dissection, and hematoma. Immediately after OCT imaging, a coronary angiogram showed a filling defect surrounded by contrast medium at the site of the stented lesion. OCT imaging was performed again and a low backscattering protrusion suggestive of white thrombus in the coronary lumen was clearly visualized in OCT imaging. We performed thrombus aspiration immediately after OCT imaging. Aspirated thrombi were off-white in color. We made a diagnosis of early-onset heparin-induced-thrombocytopenia (HIT) due to thrombus formation within the stent and positive HIT antibodies. OCT in the acute phase of stent thrombosis allowed us to promptly identify the main causative mechanisms of early stent thrombosis. (Int Heart J 2016; 57: 763-765)

Key words: Intravascular imaging, Acute myocardial infarction, Coronary stent

Heparin-induced thrombocytopenia (HIT) is a rare but critical problem in daily clinical practice, especially during treatment with percutaneous coronary intervention (PCI). Intracoronary thrombus is clearly visualized by optical coherence tomography (OCT) and OCT can differentiate white thrombus from red thrombus. However, coronary lesions with HIT induced thrombosis have not been well characterized. Therefore, we report here a patient with HIT, whose local thrombosis was assessed with OCT and histopathology.

Case Report

A 62-year-old man with a family history of coronary artery disease and a history of smoking, diabetes, and dyslipidemia was admitted to our hospital with chest pain from acute myocardial infarction. He was given aspirin (200 mg), clopidogrel (300 mg), and an unfractionated heparin (UFH 5000 unit) loading dose prior to the emergent coronary angiography. Optical coherence tomography was performed with intervention to a mid-right coronary occlusion with drug-eluting stent implantation. OCT (Dragonfly OPTIS, St. Jude Medical, Minneapolis, MN) visualized well-apposed stent struts and no remarkable tissue protrusion, stent underexpansion, malapposition, edge dissection, and hematoma. Immediately after OCT imaging, a coronary angiogram showed a filling defect surrounded by contrast medium at the site of the stented lesion (upper left). OCT imaging was performed again and a low backscattering protrusion suggestive of white thrombus in the coronary lumen was clearly visualized in OCT imaging (upper right). We performed thrombus aspiration immediately after OCT imaging. Aspirated thrombi were off-white in color. The activated clotting time (ACT) was 243 seconds at that time. We doubted the thrombi was caused by heparin-induced-thrombocytopenia (HIT) since there was no evidence of edge dissection, stent malapposition, or underexpansion. Approximately one hour passed after 5000 units of heparin injection. Shortly thereafter, we administered the direct thrombin inhibitor argatroban instead of using heparin. Urokinase (120,000 IU) through the catheter was infused and coronary flow was improved to thrombolysis in acute myocardial infarction (TIMI) 3 from TIMI 2. Although this patient had not previously been exposed to any kind of heparin, we diagnosed early-onset HIT due to thrombus formation within the stent and positive HIT antibodies (1.0 U/mL). The histology for aspirated thrombus was a platelet-based white thrombus. After the procedure, the platelet count was not decreased and this patient was discharged 2 weeks later without a thrombotic event. After 2 months, a VerifyNow P2Y12 Assay (Accumetrics, San Diego, CA) for mea-
uring the responsiveness to clopidogrel showed no resistance to clopidogrel (P2Y12 reaction units: 131).

**DISCUSSION**

Heparin-induced-thrombocytopenia (HIT) is an immunological mechanism involving the formation of complexes between exogenous heparin from animal sources and antibodies to platelet factor 4 (PF4)-heparin complexes. Preexisting HIT antibodies are detected in some patients who have not previously been exposed to any kind of heparin. A previous study reported that preexisting HIT antibodies were more common in acute coronary syndrome (ACS) patients than in non-ACS patients. Therefore, we believe that our ACS case with preexisting HIT antibodies developed thrombotic complications during PCI even in such a short time. VerifyNow P2Y12 measurement was performed 2 months after PCI and showed no resistance to clopidogrel. However, the exact relationship between resistance to clopidogrel and stent thrombosis at PCI procedure is unclear.

Using OCT in the acute phase of stent thrombosis allows us to promptly identify the main causative mechanisms of early stent thrombosis, such as stent underexpansion, malapposition, edge dissection, hematoma, and tissue protrusion. HIT should be suspected if OCT visualizes a new white thrombus just after stent implantation without evidence of the above coronary flow limiting problems that may cause early stent thrombosis.

**DISCLOSURE**

Conflict of interest: There is no conflict of interest to declare.

**REFERENCES**


Figure. Optical coherence tomography (OCT) findings in lesion with early stent thrombosis. Angiographic (left) and optical coherence tomography (OCT) findings (right) in lesion with early stent thrombosis and corresponding histology (bottom) for aspirated white thrombus.