Intracoronary thrombus formation on a guide wire: who is to blame?

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Case report: A 77-year-old man with stable angina and known left main coronary artery (LMCA) disease was admitted for elective percutaneous coronary intervention (PCI). Dual antiplatelet therapy with aspirin and clopidogrel had been instituted 7 days before the PCI, accompanied by administration of unfractionated heparin (UHF) during the procedure. For the PCI planning purpose, intravascular ultrasound was performed to evaluate the anatomy and the size of the LMCA and the morphology of the left anterior descending artery-first diagonal (LAD-D1) bifurcation lesion that had appeared only moderately stenotic on angiography. Minimal lumen diameter of the middle LAD segment and LMCA was 1.6 mm and 2.6 mm, respectively, indicating the severity of both lesions. After predilatation with the semicompliant balloon, a successful provisional stenting of the LAD-D1 bifurcation lesion using drug-eluting stent (DES) was performed, followed by high-pressure postdilatation to optimize stent deployment. At that time, the thrombus formation on a guide wire within the LMCA was noted, giving rise to distal LAD embolization. Direct stenting of the proximal LMCA with DES 4.0/9 mm was then performed and the intracoronary bolus of eptifibatide was given, leading to complete LMCA thrombus resolution, with residual thrombi seen in the distal LAD segments. Due to chest pain aggravation, a second-look angiography was performed 2 hours after the index procedure, showing non-occlusive dissection at the distal end of the LMCA stent. Additional DES 4.0/9 mm was implanted with optimal result and Thrombolysis In Myocardial Infarction 3 flow. Clopidogrel was replaced with ticagrelor. The 6-month follow-up was uneventful.

Discussion: Pharmacotherapy during PCI is used to mitigate the sequel of iatrogenic plaque rupture and to reduce the risk of thrombus formation on intravascular PCI equipment. Iatrogenic damage to the endothelium leads to increased expression of tissue factor and activation of the coagulation cascade, ultimately leading to thrombus formation[1]. Anticoagulation with UFH alone does not seem to be sufficient for protection from ischemic sequel, such as periprocedural myocardial infarction. One cause of these events is embolization of platelet aggregates that form as a result of platelet activation induced by UFH[2] and the prevention of thrombus formation strongly depends on platelet inhibition by dual antiplatelet therapy. In cases with high-risk plaque features, a care has to be taken to recognize iatrogenic damage in a timely manner. Usefulness of clopidogrel resistance testing before complex PCI has yet to be shown.

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