

Repetitive stent thrombosis in a patient with suspected allergy to aspirin and multiple switch between clopidogrel, prasugrel, and ticagrelor

Nawracająca zakrzepica w stencie u chorego z podejrzeniem alergii na kwas acetylosalicylowy i z wielokrotną zmianą leczenia przeciwplatekowego

Bogumił Ramotowski, Anna Żuk, Andrzej Budaj

Department of Cardiology, Postgraduate Medical School, Grochowski Hospital, Warsaw, Poland

Acute stent thrombosis is a rare but potentially fatal complication of percutaneous coronary intervention (PCI). Among the most important risk factors for stent thrombosis are premature discontinuation of antiplatelet treatment, the use of single antiplatelet therapy, and procedural factors such as incomplete stent apposition or residual dissection. New antiplatelet agents reduce the risk of stent thrombosis, but switching between ticagrelor and prasugrel may increase platelet reactivity in the first 48 h. We present an illustrative case of a 59-year-old male admitted to our hospital due to 1.5-h chest pain with electrocardiographic (ECG) signs of anterolateral ST-elevation myocardial infarction (STEMI). The patient was a smoker (10 cigarettes/day) with history of hypertension, hypercholesterolaemia, and asthma. The patient declared an allergy to acetylsalicylic acid (ASA), so single antiplatelet therapy with clopidogrel was administered. Emergency coronary angiography demonstrated occluded left anterior descending artery (LAD) (Fig. 1), which was treated with PCI with deployment of two overlapping drug eluting stents (DES) (Fig. 2). The history of ASA allergy turned out to be unclear. Therefore, there was an attempt to administer ASA complicated by bronchospastic reaction. Because of the complex procedure, single antiplatelet therapy with clopidogrel was switched to ticagrelor. After eight days of favourable clinical course, the patient developed acute chest pain with new signs of anterolateral STEMI. Emergency coronary angiography revealed stent thrombosis in LAD (Fig. 3). After abciximab infusion and thrombectomy the dissection in the distal edge of the stent was visualised (Fig. 4), followed by deployment of another DES. Due to thrombosis during ticagrelor treatment, the antiplatelet therapy was switched to prasugrel with a loading dose of 60 mg and 10 mg daily. Three days later another acute chest pain with ECG signs of anterolateral STEMI occurred, with the recurrence of stent thrombosis in LAD. The procedure was completed with thrombectomy, abciximab infusion, and plain old balloon angioplasty (POBA). After stent thrombosis on prasugrel the antiplatelet therapy was again switched to ticagrelor. ASA intolerance was verified, and coincident interruption of ordinary asthma treatment was revealed as a likely cause of bronchospastic reaction. Consecutive ASA administration did not provoke any bronchospastic symptoms. The platelet reactivity VerifyNow test, performed due to use of abciximab after 14 days, revealed a very good effect of ticagrelor — 8 PRU (P2Y12 reaction units) and a modest effect of ASA — 580 ARU (aspirin reaction units). This case illustrates that stent thrombosis may affect patients treated with novel antiplatelet agents due to residual dissection or stent malapposition. A history of ASA allergy requires careful investigation in each case. Switching between ticagrelor and prasugrel, associated with increased platelet reactivity reported in the literature, should be avoided or carefully controlled during hospitalisation.



Figure 1. Patient with anterolateral ST elevation myocardial infarction — occluded left anterior descending artery (LAD)



Figure 2. Patient after successful percutaneous coronary intervention left anterior descending artery (LAD) with two drug-eluting stents in LAD

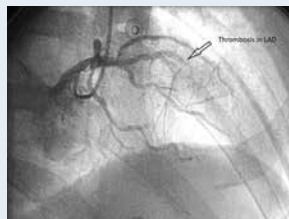


Figure 3. Acute in-stent thrombosis in left anterior descending artery (LAD)



Figure 4. Thrombosis in left anterior descending artery (LAD) after plain old balloon angioplasty with visible dissection in distal LAD

Address for correspondence:

Bogumił Ramotowski, MD, Department of Cardiology, Postgraduate Medical School, Grochowski Hospital, ul. Grenadierów 51/59, 04-073 Warszawa, Poland, e-mail: bram@onet.pl

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