Kardiologia Polska 2016; 74, 7: 699; DOI: 10.5603/KP.2016.0102 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Identifying culprit lesions in non-ST-elevation myocardial infarction: are new diagnostic tools always better?

Identyfikacja zmian angiograficznych odpowiedzialnych za zawał serca bez uniesienia odcinka ST: czy nowe narzędzia diagnostyczne zawsze są lepsze?

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A 55-year-old male was admitted to the Coronary Care Unit due to chest discomfort that started 10 days previously. Retrosternal pain was present at rest and gradually increased in intensity. In his medical record the patient had hypertension, was an ex-smoker, and had a family history of ischaemic heart disease. In the initial electrocardiogram (ECG) the occurrence of negative T waves in V2–V4 leads was observed. Enzyme profile of myocardial necrosis was slightly elevated (CK 121 IU/L, CK-MB 15.4…25.1 IU/L, troponin I 0.044… 0.046… 0.112 μg/L, upper limit normal 0.040 μg/L). Echocardiography revealed preserved left ventricular (LV) function without deterioration of segmental wall kinetics. Coronary angiography was performed within 48 h and the following findings were obtained: in the mid part of the left anterior descending artery (LAD) the stenosis was up to 50% and lower distal mild muscular bridge, left circumflex artery (LCX) with distal stenosis up to 75%, right coronary artery was dominant, with medial stenosis up to 40% (Fig. 1). Since we could not identify the culprit lesion we decided to perform additional diagnostics of LAD and LCX stenosis. The ILUMIEN optical coherence tomography (OCT) system monorail C7 Dragonfly Intravascular Imaging Catheter was delivered through a 6 F guide over a Runthrough intracoronary guidewire. OCT exploration of LAD and LCX consecutively revealed that both lesions were smooth, with border significance, and we decided not to treat them (Fig. 2). Since we were not able to identify unstable plaque, we decided to perform fractional flow reserve (FFR) measurements, aiming to determine the functional significance of this stenosis. After i.c. bolus of adenosine at a dose of 200 μg in left coronary artery (LCA), an FFR value of 0.85 in the LAD and 0.75 in the LCX was observed (Fig. 3). Direct stenting of LCX with BMS Prokinetic Energy 3.0 mm × 15 mm at 16 atm was performed. Control angiography showed good expansion of the stent. Coronary spasm was present in the whole system of the LCA and especially the site of the lesion in the LAD. Following the administration of 150 μg i.c. nitroglycerin and after Runtrough guide wire from LCX was removed, the spasm disappeared. During the subsequent days, the patient had permanent anginal complaints that were lower in intensity, without ECG changes. Four days after percutaneous coronary intervention (PCI), intensive chest was accompanied by changes in ECG in the form of new ST depression in anterior and lateral leads (↓ST; 0.5–1.5 mV) in D1, D2, AVL, V2–V6. Echocardiography was performed promptly; new deterioration in segmental wall kinetics corresponding to LAD irrigation system were spotted. Due to new circumstances, PCI of LAD was performed with primo-implantation of a drug-eluting stent Nobory 3.5 mm × 28 mm at 12 atm. During further hospitalisation and on regular monthly, six-monthly, and annual controls the patient was without problems with good toleration of exercise. In conclusion, despite the availability of sophisticated and expensive diagnostic tools such as OCT and FFR, we cannot always reliably determine the culprit lesion and perform adequate procedures. We must emphasise the role of adequate patient monitoring. ECG and echocardiography are routine, widely available, and inexpensive methods and they continue to have a significant place in the diagnosis of culprit lesions.

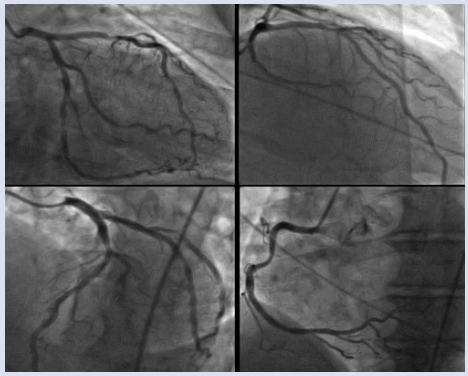

Figure 1. Coronary angiography

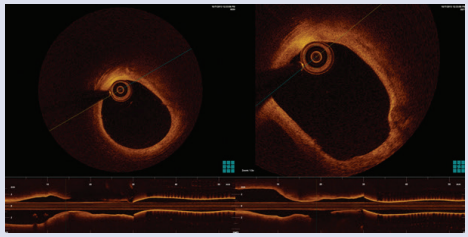

Figure 2. Optical coherence tomography exploration of vessel structure revealed smooth lesions in LAD and LCX

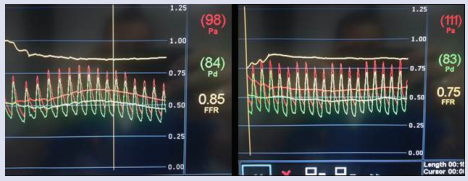

Figure 3. Fractional flow reserve measurement performed in LAD and LCX

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Conflict of interest: *none declared*