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STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Vasa vasorum-induced LAD dissection and haematoma in an anterior STEMI patient with nearly normal angiography: the role of OCT

Rozwarstwienie i krwiak śródścienny LAD przyczyną STEMI u chorego bez istotnych zmian w tętnicach wieńcowych — znaczenie OCT

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A 40-year-old male, without cardiovascular risk factors, was referred to our unit from another hospital at 4 h after anterolateral ST-segment elevation myocardial infarction (STEMI) onset (1 mm in DI and aVL and 2 mm in V2–V4) (Fig. 1-B1). Severe anterolateral hypokinesia was present on echocardiography. Cardiac necrosis was confirmed by troponin I (TnI) increase (7.8 ng/mL), and later by peak creatinine kinase (CK; 762 IU/L) and CK-MB (63 IU/L). Emergency coronary angiography was performed. Left anterior descending artery (LAD) flow was TIMI 1–2 at first injection and TIMI 3 thereafter, with normal coronary walls (Fig. 1-A1, A2). LAD imaging, using optical frequency domain imaging (optical coherence tomography [OCT]) (St. Jude Medical, St. Paul, Minnesota, USA), was performed, revealing a proximal LAD plaque with intra-plaque dissection and haematoma, induced by vasa vasorum haemorrhage and possibly stabilised by intimal tear decompression (Fig. 1C, D, E). Conservative treatment was decided upon. Electrocardiogram (ECG) evolution suggested reperfusion (Fig. 1-B2). The ECG later normalised (Fig. 1-B3). The patient was discharged 7 days later and has remained asymptomatic for the past 12 months. Coronary artery dissection or haematoma were previously correlated to important angiographic abnormalities, while our case presented with nearly normal angiography, except for the LAD flow impairment at the first injection. Before angiography and in-wall haematoma decompression, flow impairment may have been worse, due to greater lumen compression. Contrast injection during angiography may have also acted as a coronary vasodilator, thus explaining the improvement of coronary flow. OCT imaging in this patient draws attention to a particular STEMI pathophysiology, namely vasa vasorum haemorrhage (Fig. 1-C1–C5). Of particular interest is the fact that OCT imaging revealed a connection between vasa vasorum, coronary dissection, intramural haematoma and, respectively, LAD lumen. Vasa vasorum traject could extend to the coronary lumen, but there is also the possibility that decompression of in-wall haematoma, through an intimal tear, may have been the mechanism of stabilisation for this patient. OCT imaging provided the diagnosis of coronary dissection and haematoma in this STEMI patient with nearly normal angiography and gave insight into the mechanisms related to the pathogenesis and, possibly, stabilisation of a coronary dissection.

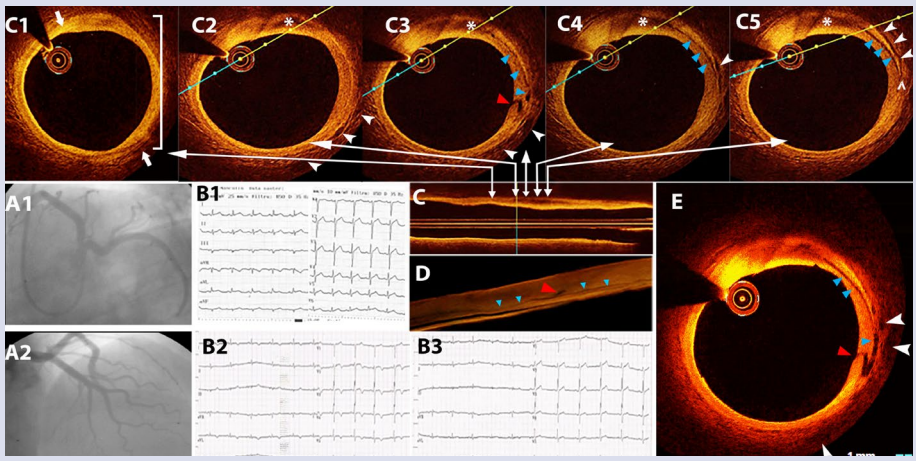

Figure 1. Proximal LAD dissection: ECG changes, coronary angiography, and OCT imaging. Angiographically nearly normal coronary arteries (**A1, A2**); ECG changes: anterior STEMI (**B1**), reperfusion signs (**B2**), ECG normalisation (**B3**); OCT imaging: an atherosclerotic plaque is present on the proximal LAD (**C1**, dotted line). Vasa vasorum (white arrowheads) are dense in the outer adventitia (**C2–C4**). A dissection line (blue arrowheads) is seen across images (**C3–C5**). A connection, on the inner adventitia, is seen between penetrating vasa vasorum and the dissection line (**C5**, open arrowhead). A connection (**C3**, red arrowhead) exists between the dissection line and the coronary lumen, through a transversal, tortuous traject. Dissection line ends in a small circumscribed haematoma (**C2–C5**, asterisk). A three-dimensional reconstruction (**D**) of the inner LAD wall identifies the intimal tear and the dissection line extending both proximally and distally. A Photoshop CC2015 blending technique of 10 consecutive OCT images (**E**) reveals connections between vasa vasorum, dissection line, and coronary lumen

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