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IMAGE

Spontaneous dissecting coronary haematoma with and without intimal tear

Hématome coronaire spontané disséquant avec et sans rupture intimale

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Acute coronary obstruction in ST-segment elevation myocardial infarction (STEMI) is not necessarily atherothrombotic: intraplaque haemorrhage and the resulting intra-adventitial haematoma may also be implicated, spreading longitudinally along the coronary artery, dissecting the tunicae, especially if the adjacent arterial segments are normal. Such dissecting haematoma may appear on the angiogram with or without associated intimal tearing. When there is no intimal tear, angiographic diagnosis is difficult, although the length, filiform and tubular aspect of the obstruction with long abrupt change in arterial diameter may be suggestive, notably in women less than 50 years of age. Signs specific to this STEMI aetiology, however, can be detected on intravascular ultrasound (IVUS) or optical coherence tomography (OCT), on which the aspect is characteristic and pathognomic.

In case of positive diagnosis, any intervention, especially with primary stenting, is to be delayed for as long as the mechanism and segment length involved preclude a good angiographic and functional result. Sometimes, angioplasty is performed using only a small balloon at low pressure (4 bars), maintained (3 min) to redistribute the haematoma until Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 is achieved. The haematoma should be allowed to achieve spontaneous resorption by reducing antiplatelets and stopping anticoagulants, with control coronary angiography at D3–5 to allow discharge at a subsequent control at 2 months to check the final evolution and correct any residual obstruction.

Abbreviations: IVUS, intravascular ultrasound; OCT, optical coherence tomography; STEMI, ST-segment elevation myocardial infarction.

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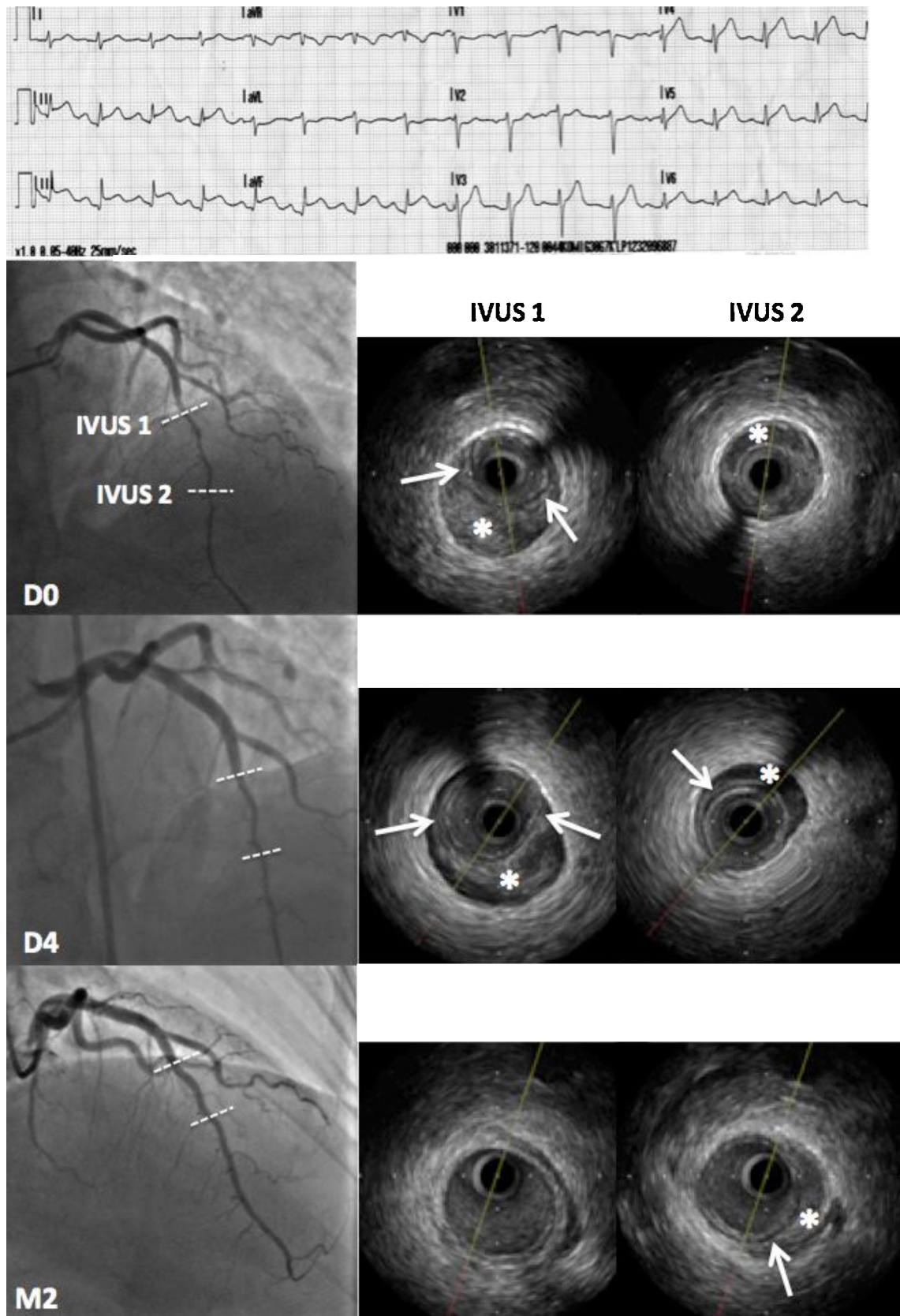


Figure 1. Spontaneous dissecting coronary haematoma without intimal tear. Long and abrupt change in left anterior descending diameter on angiography. Characteristic signs of intra-advntial haematoma on intravascular ultrasound (IVUS) (*), compressing the normal coronary artery with its typical three-layer appearance (white arrows). Over spontaneous evolution (D4 and M2), the haematoma alters in structure on ultrasound, almost completely resorbing, and the lumen cross-section increases in area to become normal.

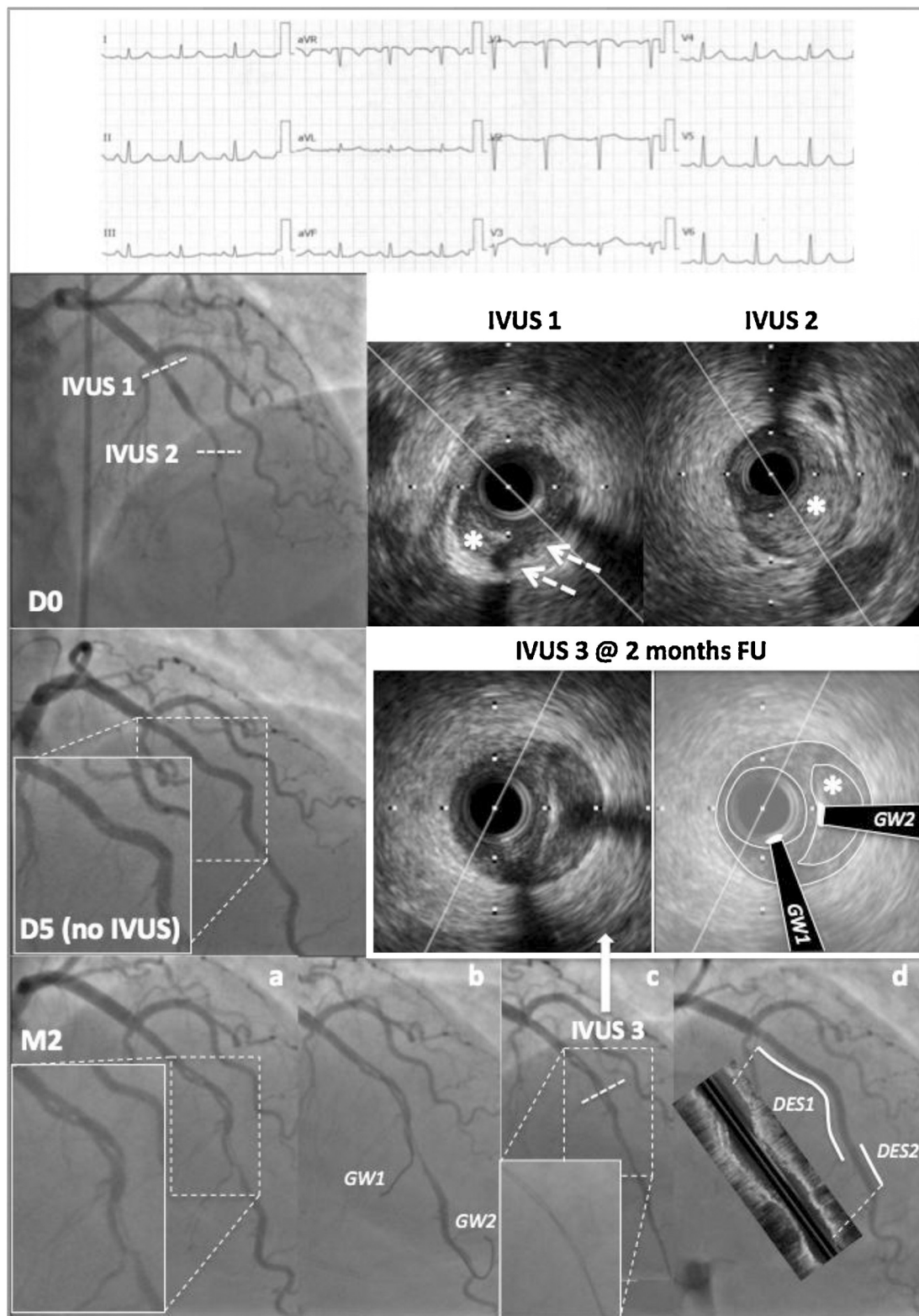


Figure 2. Spontaneous dissecting coronary haematoma with intimal tear. Long and abrupt change in left anterior descending diameter with subocclusion on angiography. Characteristic signs of intra-adventitial haematoma on intravascular ultrasound (IVUS) (*), with intimal tear (IVUS 1, dotted white arrows). At D5, persisting aspect of intimal dissection associated with increased lumen calibre (no IVUS performed, by precaution). At M2, a: persisting obstructive (90%) spiroid dissection; b: two guidewires (GW1 and GW2) introduced in either channel in two different side branches; c: optimal replacement of the two guidewires in each channel and precise determination of the true channel on IVUS (GW1), with GW2 in the false channel (*); d: precise optimal implantation of two overlapping drug-eluting stents (DES) to treat the 40mm obstruction. FU: follow-up.

We report two cases of STEMI, illustrating the process of diagnosing dissecting haematoma, with and without intimal tear, and their evolutions (Figs. 1 and 2). Both forms were induced by the same haemorrhagic mechanism: haematoma, lacerating the coronary arterial wall, complicating nascent atherosclerosis. When such a mechanism is detected, as it should be on IVUS or OCT, the appropriate approach should not be systematic stenting but rather to allow spontaneous

resorption by reducing antiplatelets and stopping anticoagulants, with invasive check-up at 2 months.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.