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CORONARY STENTING AFTER FAILURE OF CONSERVATIVE TREATMENT FOR SPONTANEOUS CORONARY DISSECTION. USEFULNESS OF THE INTRAVASCULAR ULTRASOUND

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**CORONARY STENTING AFTER FAILURE OF CONSERVATIVE
TREATMENT
FOR SPONTANEOUS CORONARY DISSECTION.
USEFULNESS OF THE INTRAVASCULAR ULTRASOUND**

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Manuscripts

Review

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CORONARY STENTING AFTER FAILURE OF CONSERVATIVE TREATMENT
FOR SPONTANEOUS CORONARY DISSECTION.
USEFULNESS OF THE INTRAVASCULAR ULTRASOUND

For Peer Review

ABSTRACT

Spontaneous coronary dissection of a coronary artery is a rare cause of acute coronary syndrome. Optimal treatment has not yet been established. We describe a woman with spontaneous dissection of the left anterior descending artery who presented transitory angina and electrocardiographic ST segment elevation in precordial leads. Conservative treatment was carried out. The angina and electrocardiographic changes recurred three days later. An intravascular ultrasound was performed and the patient was stabilized following stenting. Stress testing was negative at six-month follow up and she remained asymptomatic at 18 months. We discuss the usefulness of an intravascular ultrasound study to establish the therapeutic strategy.

INTRODUCTION

Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome and sudden death. Prevalence is highest in the peripartum or postpartum period in young women with no cardiovascular risk. It has occasionally been related to intensive physical exercise, cocaine abuse, collagen vascular diseases and antiphospholipidic syndrome. Spontaneous coronary artery dissection most commonly affects the left main coronary artery, the left anterior descending coronary artery (LAD), or both. Prognosis is related to early diagnosis^{1,2}. We describe a woman who had spontaneous coronary artery dissection of the LAD artery in whom angina recurred after initial conservative treatment. She was successfully treated with stent implantation. We emphasise the usefulness of the intravascular ultrasound study (IVUS) before stent implantation.

CASE REPORT

A 52-year-old woman was referred to our coronary care unit with intensive chest pain during moderate effort, associated with autonomic symptoms of 20 minutes' duration, and an ECG showing ST segment elevation of 2 mm in v_2 to v_4 precordial leads. She had a history of smoking, mild obesity, and arterial hypertension treated with bisoprolol. She had recently lost 5 kg in about two weeks as the result of a rigorous hyperproteic diet. When the patient arrived at our center she was asymptomatic and the electrocardiogram was normal. The coronary angiogram showed a diffuse luminal narrowing of the LAD artery from below the origin of the first diagonal to the apical segment (Figure 1, A). Diffuse coronary dissection was diagnosed in the LAD artery. The right coronary and circumflex arteries were normal. Creatine phosphokinase was 142 U/L and troponin was T 0.118 $\mu\text{g/L}$. Conservative treatment (atenolol, nitroglycerine, clopidogrel, acetyl-salicylic acid, atorvastatin and intermediate doses of enoxaparin) was adopted because the patient was stable. Three days later, she presented a recurrent episode of angina with ST elevation of 2 mm, extending to leads v_5 - v_6 . A second coronary angiogram showed persistence of the diffuse narrowing of the

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3 distal LAD artery and occlusion at the apical segment (Figure 2). Before performing an
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5 IVUS, we administered bivalirudin at doses of 0.75 mg/kg/weight followed by a
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7 perfusion of 1.5 mg/kg during the procedure. After placing an angioplasty catheter and
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9 passing a 0.014" guide-wire to the LAD artery, an IVUS using an Atlantis SR Pro 40
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11 MHz coronary imaging catheter (Boston Scientific, Freemont, Ca) was performed to
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13 establish correct positioning of the guide-wire in the true lumen and to assess the
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15 extension of dissection. No hematoma was seen in the left main or in the proximal or
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17 medium segments of the LAD artery (Figure 2, slice 1). A large hypodense image
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19 observed about 3 mm below the origin of the diagonal artery was suggestive of an
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21 intramural hematoma likely due to recent bleeding (Figure 2, slice 2). A dense extra-
22
23 luminal image suggested an organized hematoma extending through the distal
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25 segment with internal hypodense areas (Figure 2, slice 3-4). Since the patient had
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27 angina and ST elevation we decided to stent the LAD artery. Stenting was started
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29 distally, progressing upwards. Six overlapping conventional stents were implanted:
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31 Vision 2,25x18 mm (Abbot Vascular, Santa Clara, Ca); Vision 2,75x12 mm; Driver
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33 2,75x18 mm (Medtronic Inc, Minneapolis, MN), Driver 3.0x24 mm, Driver 3x18 mm,
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35 Driver 3.25x30 mm. Balloons were inflated to 15-20 atm, including the overlapping
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37 stent segments and flow was reestablished (Figure 1,B). The chest pain disappeared
38
39 and the ECG normalized. Creatine phosphokinase was 807 U/L and T troponin was 1.8
40
41 µg/L. Echography showed mild hypokinesis with a global left ventricular ejection
42
43 fraction of 60%. Laboratory tests were performed to rule out associated connective
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45 tissue disorders and vasculitis. No etiologic cause of the dissection was found. At
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47 discharge, in addition to oral nitroglycerin and atenolol, we prescribed clopidogrel 75
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49 mg/day for one year and acetyl-salicylic acid 100 mg/day indefinitely. At 6-month
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51 follow-up the myocardial perfusion study using a photon emission computed
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53 tomography (SPECT) showed a focal apical necrosis without signs of ischemia in the
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55 remaining segments. At 18-month follow up the patient was free of ischemic symptoms
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57 and a conventional stress test was negative.
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DISCUSSION

The pathogenesis of spontaneous coronary artery dissection is related to a coronary artery disruption that causes a false lumen with an intramural hemorrhage. It progressively compresses the true lumen, leading to myocardial ischemia. Diagnosis is usually made by coronary angiography in the setting of an acute coronary syndrome.

Due to the rarity of this event, there is no consensus and optimal treatment remains a challenge. Treatment should be individualised, taking into account the clinical situation, dissection extension, degree of luminal compression, myocardium at risk, multivessel or left main artery involvement and underlying atherosclerotic disease. Conservative treatment with beta-blockers, antiplatelet aggregation and anticoagulation may resolve the hematoma and prevent thrombosis¹. This approach would be indicated in stable patients.

Optional therapies include coronary artery bypass and stenting. Patients with underlying atherosclerotic disease, multiple vessel dissection or involvement of the left main artery could benefit from an aortocoronary bypass³⁻⁵. Coronary stenting is an increasingly used approach as it rapidly provides lumen permeability^{4,5}. It is a valid option in unstable patients with ST elevation with angina and in stable patients with a focal dissection. Stenting has also been successful in patients with left main artery involvement⁶ and multi-vessel disease⁷. The inconvenience of stenting is that hematoma may spread, increasing the need for further stenting⁸. To avoid this complication in the present patient we implanted the stents in an upward direction, from the distal to the proximal segment as far as the healthy arterial segment. Stenting may also cause occlusion of the lateral branch due to the adventitial distribution of the hematoma in the arterial wall. Late complications of stenting are restenosis and lack of stent apposition once the hematoma has been reabsorbed, especially when small diameter stents have been implanted. Use of bare-metal and drug-eluting stents has been reported^{5,9} but the restenosis rate is unknown. Since our patient was asymptomatic and spontaneous reabsorption of the hematoma was likely, conservative

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3 treatment was decided to avoid the need for cardiac surgery or multiple stenting.
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5 However, three days later the ST segment again became elevated and stent
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7 implantation was required. We decided to implant bare-metal stents instead of drug-
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9 eluting stents in view of the positive results reported in the literature and because of the
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11 risk of later thrombosis with drug-eluting stents^{5,9,10}. IVUS was useful to plan the
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13 therapeutic strategy. It differentiated luminal narrowing due to intramural hematoma
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15 from a spasm or a diffuse atherosclerotic lesion. It allowed us to assess the extension
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17 of the dissection and helped to clarify whether the left main and the proximal LDA
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19 artery were also involved, thereby limiting stent implantation. Since coronary
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21 angiography provides only a luminal image, stent size calculation is difficult, especially
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23 in long dissections. IVUS in this setting allows accurate calculation of vessel size. In
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25 coronary dissection there is a risk of inadvertently wiring the false lumen. IVUS assures
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27 that the guide-wire is placed in the true lumen, yielding safe stent implantation. In the
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29 present case, IVUS helped us to localize the entrance towards the false lumen and to
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31 understand the mechanism of recurrence of angina. Distal LAD artery occlusion was
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33 seen at the apical level in the second coronary angiogram. IVUS suggested that the
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35 entrance was a few millimeters beyond the origin of the first diagonal artery and that
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37 occlusion of the distal LAD may have been responsible for the sudden re-bleeding into
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39 the false lumen on the third day, causing downwards expansion of the hematoma. This
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41 possibility indicates that close surveillance is needed in patients receiving conservative
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43 treatment.
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49 In summary, the present case describes the failure of conservative therapy in a woman
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51 with spontaneous coronary dissection, highlighting the relevance of IVUS in diagnosis,
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53 and illustrating the good results with stent implantation.
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8 **Figure 1. A:** The coronary angiogram showed a diffuse luminal narrowing of the
9 anterior descending coronary artery from close to the first diagonal to the apical
10 segment due to spontaneous coronary dissection (arrows). **B:** Coronary angiogram
11 after stenting.
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20 **Figure 2. Above:** Angiogram showing occlusion of the apical segment of the anterior
21 descending coronary artery. Numbered cross-sectional intravascular ultrasound
22 images and the corresponding transversal segment in the coronary angiogram of the
23 anterior left descending coronary artery are shown. **Below:** Four ultrasound images are
24 shown. Slice 1 shows an ultrasound image with a small atherosclerotic plaque in the
25 arterial wall and the origin of the first diagonal branch. No image of dissection is seen.
26
27 Slice 2. Note the hypodense images suggestive of recent bleeding. Slice 3. Image of
28 an organized hematoma encircling almost the entire circumference of the vessel. Slice
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30 Slice 4. Image of an organized hematoma with internal hyperdense areas of unclotted blood.
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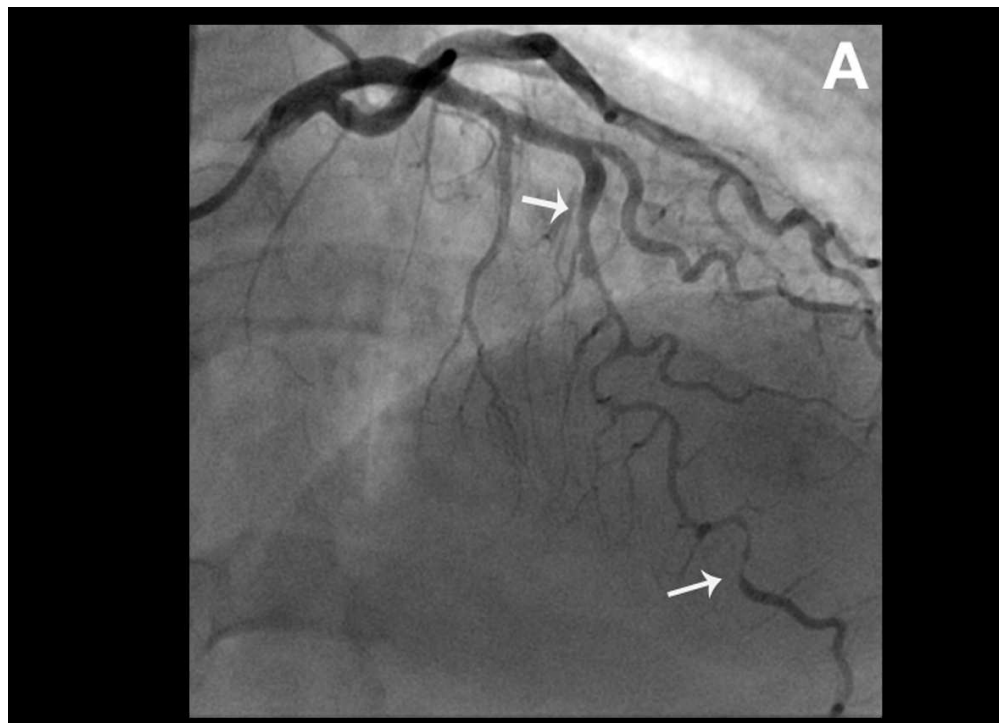
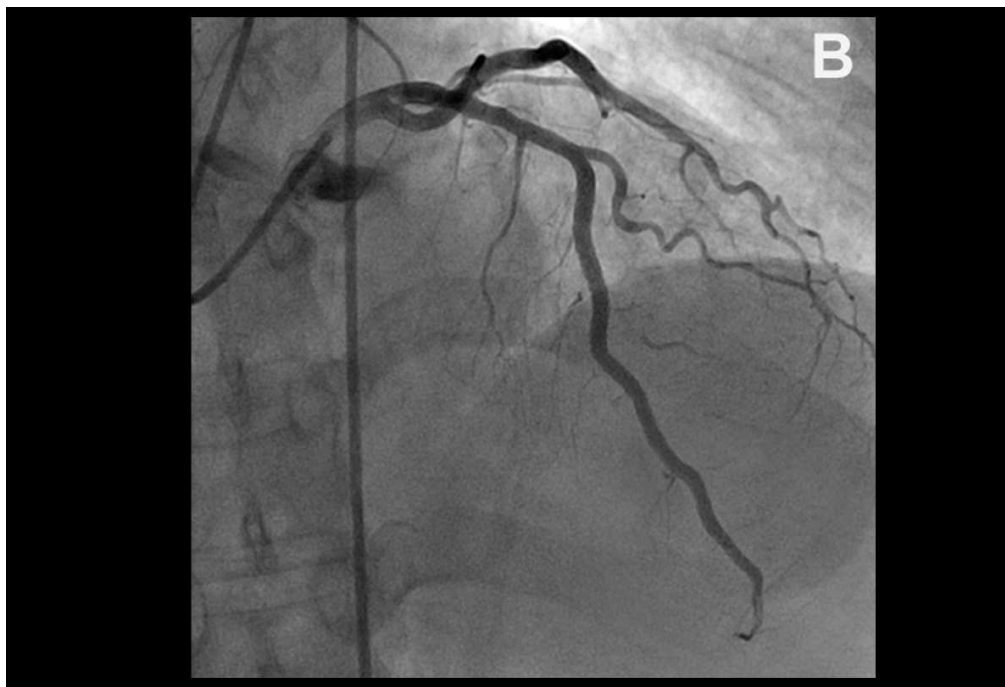


Figure 1. A: The coronary angiogram showed a diffuse luminal narrowing of the anterior descending coronary artery from close to the first diagonal to the apical segment due to spontaneous coronary dissection (arrows).

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B: Coronary angiogram after stenting.

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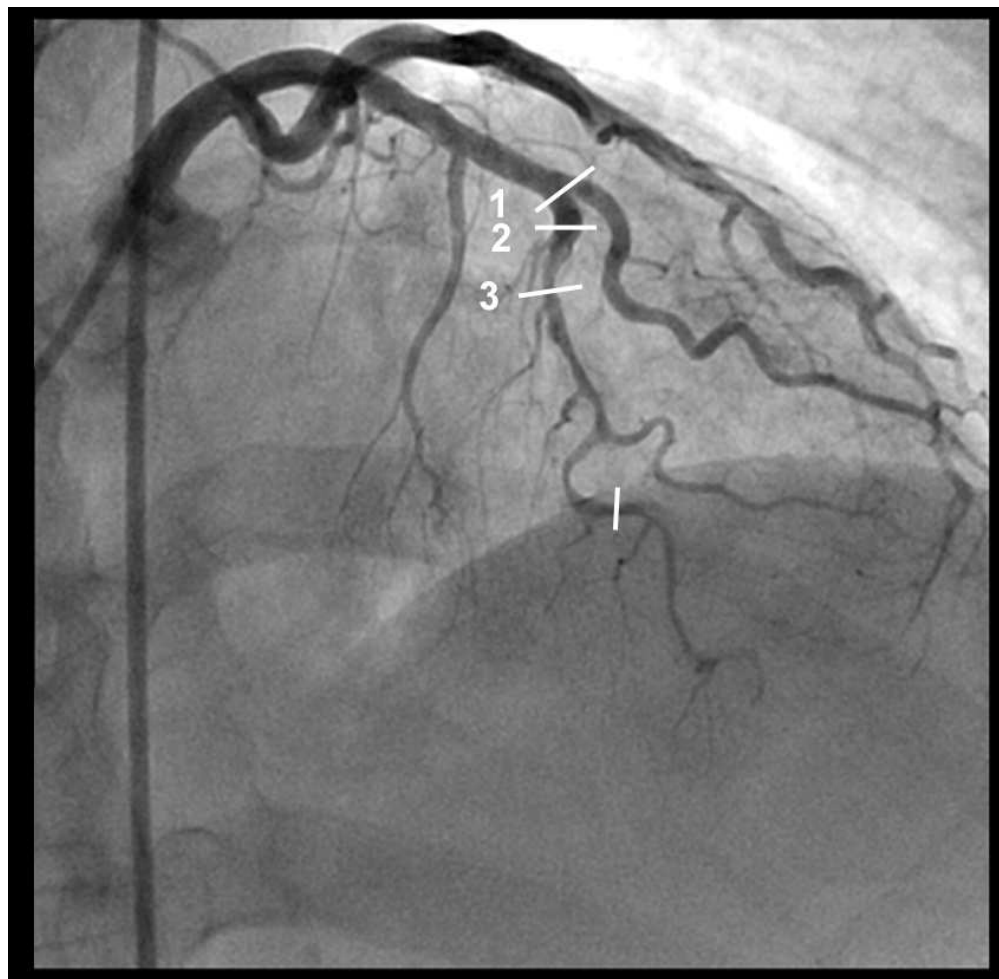
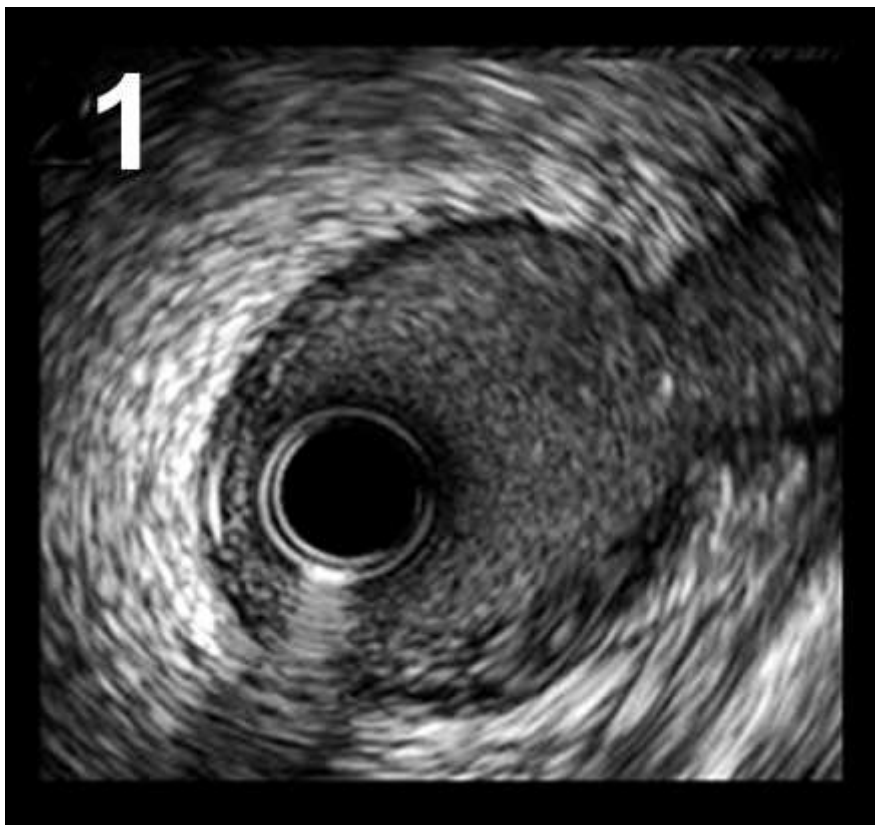


Figure 2. Above: Angiogram showing occlusion of the apical segment of the anterior descending coronary artery. Numbered cross-sectional intravascular ultrasound images and the corresponding transversal segment in the coronary angiogram of the anterior left descending coronary artery are shown.

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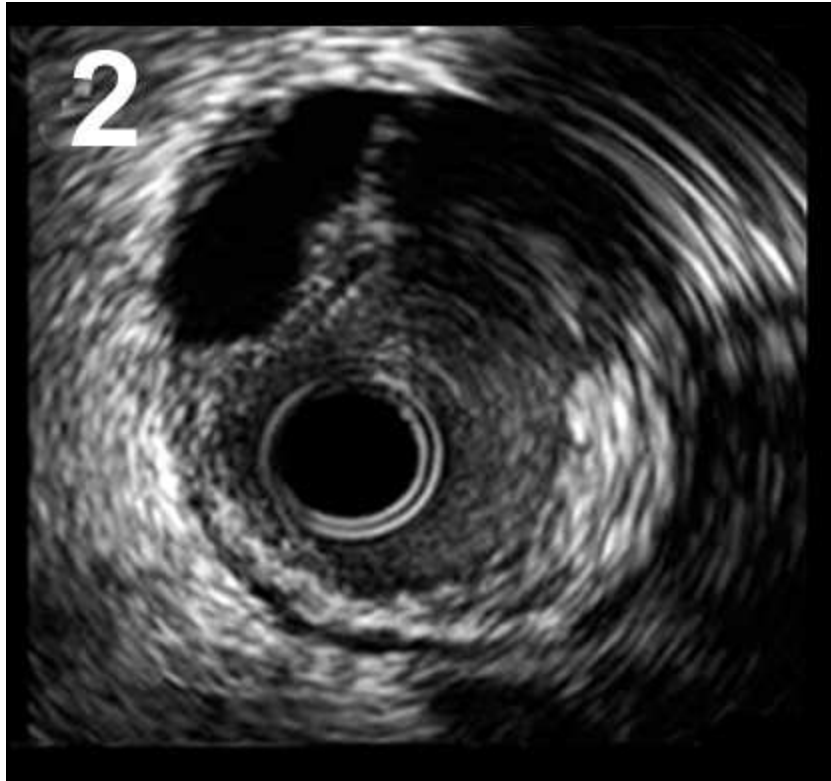
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Below: Four ultrasound images are shown. Slice 1 shows an ultrasound image with a small atherosclerotic plaque in the arterial wall and the origin of the first diagonal branch. No image of dissection is seen.

view

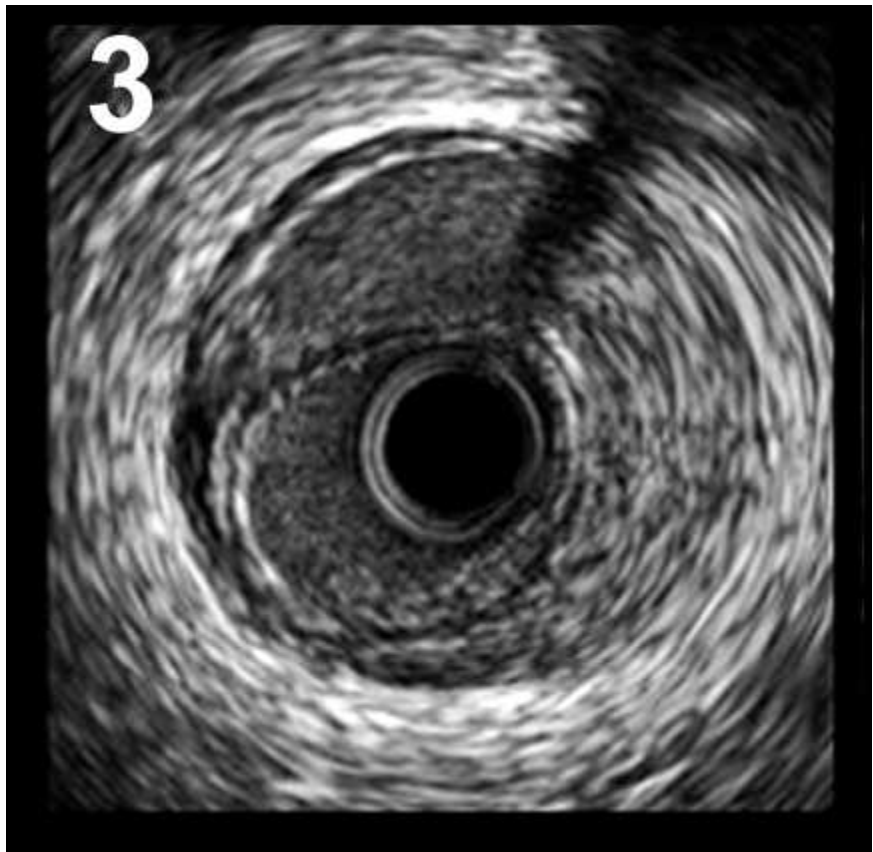
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Slice 2. Note the hypodense images suggestive of recent bleeding.

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Slice 3. Image of an organized hematoma encircling almost the entire circumference of the vessel.

view



Slice 4. Image of an organized hematoma with internal hyperdense areas of unclotted blood.

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CORONARY STENTING AFTER FAILURE OF CONSERVATIVE TREATMENT
FOR SPONTANEOUS CORONARY DISSECTION.
USEFULNESS OF THE INTRAVASCULAR ULTRASOUND ECHOCARDIOGRAPHY
STUDY

For Peer Review

ABSTRACT

Spontaneous coronary dissection of a coronary artery is a rare cause of acute coronary syndrome. Optimal treatment has not yet been established. We describe a woman with spontaneous dissection of the left anterior descending artery who presented transitory angina and electrocardiographic ST segment elevation in precordial leads. Conservative treatment was carried out. The angina and electrocardiographic changes recurred three days later. An intravascular ultrasound was performed and the patient was stabilized following stenting. Stress testing was negative at six-month follow up and she remained asymptomatic at ~~40~~ 18 months. We discuss the usefulness of an intravascular ultrasound study to establish the therapeutic strategy.

INTRODUCTION

Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome and sudden death. Prevalence is highest in the peripartum or postpartum period in young women with no cardiovascular risk. It has occasionally been related to intensive physical exercise, cocaine abuse, collagen vascular diseases and antiphospholipidic syndrome. Spontaneous coronary artery dissection most commonly affects the left main coronary artery, the left anterior descending coronary artery (LDA), or both. Prognosis is related to early diagnosis^{1,2}. We describe a woman who had spontaneous coronary artery dissection of the LAD artery in whom angina recurred after initial conservative treatment. She was successfully treated with stent implantation. We emphasise the usefulness of the intravascular ultrasound study (IVUS) before stent implantation.

CASE REPORT

A 52-year-old female was referred to our coronary care unit with intensive chest pain during moderate effort, associated with ~~autonomic symptoms~~ ~~vegetatism~~ of 20 minutes' duration, and an ECG showing ST segment elevation of 2 mm in v_2 to v_4 precordial leads. She had a history of smoking, mild obesity, and arterial hypertension treated with bisoprolol. She had recently lost 5 kg in about two weeks as the result of a rigorous hyperproteic diet. When the patient arrived at our center she was asymptomatic and the electrocardiogram was normal. The coronary angiogram showed a diffuse luminal narrowing of the LAD artery from below the origin of the first diagonal to the apical segment (Figure 1, A). Diffuse coronary dissection was diagnosed in the LAD artery. The right coronary and circumflex arteries were normal. Creatine phosphokinase was 142 U/L and troponin was T 0.118 μ g/L. Conservative treatment (atenolol, nitroglycerine, clopidogrel, acetyl-salicylic acid, ~~elxane~~ ~~and~~ atorvastatine and intermediate doses of enoxaparin) was adopted because the patient was stable. Three days later, she presented a recurrent episode of angina with ST elevation of 2 mm, extending to leads v_5 - v_6 . ~~High doses of morphine were needed to alleviate the~~

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2 ~~pain.~~ A second coronary angiogram showed persistence of the diffuse narrowing of the
3 distal LAD artery and occlusion at the apical segment (Figure 2). Before performing an
4 IVUS, we administered bivalirudin at doses of 0.75 mg/kg/weight followed by a
5 perfusion of 1.5 mg/kg during the procedure. After placing an angioplasty catheter and
6 passing a 0.014" guide-wire to the LAD artery, an IVUS using an Atlantis SR Pro 40
7 MHz coronary imaging catheter (Boston Scientific, Fremont, Ca) was performed to
8 establish correct positioning of the guide-wire in the true lumen and to assess the
9 extension of dissection. No hematoma was seen in the left main or in the proximal or
10 medium segments of the LAD artery (Figure 2, slice 1). A large hypodense image
11 observed about 3 mm below the origin of the diagonal artery was suggestive of an
12 intramural hematoma likely due to recent bleeding (Figure 2, slice 2). A dense extra-
13 luminal image suggested an organized hematoma extending through the distal
14 segment with internal hypodense areas (Figure 2, slice 3-4). Since the patient had
15 angina and ST elevation ~~we decided~~ ~~it was decided~~ to stent the LAD artery. Stenting
16 was started distally, progressing upwards. Six overlapping conventional stents were
17 implanted: Vision 2,25x18 mm (Abbot Vascular, Santa Clara, Ca); Vision 2,75x12 mm;
18 Driver 2,75x18 mm (Medtronic Inc, Minneapolis, MN), Driver 3.0x24 mm, Driver 3x18
19 mm, Driver 3.25x30 mm. Balloons were inflated to 15-20 atm, including the overlapping
20 stent segments and flow was reestablished (Figure 1,B). The chest pain disappeared
21 and the ECG normalized. Creatine phosphokinase was 807 U/L and T troponin was 1.8
22 µg/L. Echography showed mild hypokinesis with a global left ventricular ejection
23 fraction of 60%. Laboratory tests were performed to rule out associated connective
24 tissue disorders and vasculitis. No etiologic cause of the dissection was found. At
25 discharge, in addition to oral nitroglycerine and atenolol, we prescribed clopidogrel 75
26 mg/day for one year and acetyl-salicylic acid 100 mg/day indefinitely. ~~A mild apical~~
27 ~~hypokinesia was observed by echocardiography with a global left ejection fraction of~~
28 ~~60%.~~ At 6-month follow-up the myocardial perfusion study using a photon emission
29 computed tomography (SPECT) showed a focal apical necrosis without signs of

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2 ischemia in the remaining segments. At ~~40-~~ 18-month follow up the patient was free of
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4 ischemic symptoms and a conventional stress test was negative.

6 DISCUSSION

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8 The pathogenesis of spontaneous coronary artery dissection is related to a coronary
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10 artery disruption that causes a false lumen with an intramural hemorrhage. It
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12 progressively compresses the true lumen, leading to myocardial ischemia. Etiology is
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14 uncertain, but mechanisms such as disruption of the vasa vasorum, eosinophilic
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16 periadventitial inflammation, focal vasculitis, cystic medial necrosis and plaque rupture
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18 disruption in patient with atherosclerosis disease have been proposed. Diagnosis is
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20 usually made by coronary angiography in the setting of an acute coronary syndrome.

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22 Due to the rarity of this event, there is no consensus and optimal treatment remains a
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24 challenge. Treatment should be individualised, taking into account the clinical situation,
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26 (e.g. persistent angina, ST elevation), angiographic dissection extension, degree of
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28 luminal compression caused by the hematoma, myocardium at risk, multivessel or left
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30 main artery involvement and underlying atherosclerotic disease. Successful C
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32 Conservative treatment with beta-blockers, antiplatelet aggregation and anticoagulation
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34 may promote resolution resolve the hematoma and prevent thrombosis¹. This approach
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36 would be indicated in stable patients.

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38 Coronary artery bypass or stenting are two optional therapies. Optional therapies
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40 include coronary artery bypass and stenting³⁻⁵. Patients with underlying atherosclerotic
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42 disease, multiple vessel dissection or involvement of the left main artery could benefit
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44 from an aortocoronary bypass. However, anastomosis of the graft on a dissected
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46 coronary artery is sometimes technically difficult³. Coronary stenting is an increasingly
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48 used approach as it rapidly provides lumen permeability^{4,5}. It is a valid option as a
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50 primary election treatment in unstable patients with ST elevation with angina and in
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52 stable patients with a focal dissection. Stenting has also been successful used
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54 successfully in patients with left main artery involvement⁶ and multi-vessel disease⁷.
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2 further stenting⁸. To avoid this complication in the present patient we implanted the
3
4 stents in an upward direction, from the distal ~~segment~~ to the proximal segment as far
5
6 as the healthy arterial segment. Stenting may also cause occlusion of the lateral
7
8 branch due to the adventitial distribution of the hematoma in the arterial wall. Late
9
10 complications of stenting are restenosis and lack of stent apposition once the
11
12 hematoma has been reabsorbed, especially when small diameter stents have been
13
14 implanted. Use of bare-metal and drug-eluting stents has been reported^{5,9} but the
15
16 restenosis rate ~~in this setting~~ is unknown. Since our patient was asymptomatic and
17
18 spontaneous reabsorption of the hematoma was likely, conservative treatment was
19
20 decided ~~so as~~ to avoid the need for cardiac surgery or multiple stenting. However,
21
22 three days later the ST segment again became elevated and stent implantation was
23
24 required. We decided to implant bare-metal stents instead of drug-eluting stents in view
25
26 of the positive results reported in the literature and because of the risk of later
27
28 thrombosis with drug-eluting stents^{5,9,10}. IVUS was useful to plan the therapeutic
29
30 strategy. It differentiated luminal narrowing due to intramural hematoma from a spasm
31
32 or a diffuse atherosclerotic lesion. It allowed us to assess the extension of the
33
34 dissection and helped to clarify whether the left main and the proximal LDA artery were
35
36 also involved, ~~thereby limiting us helping to limit~~ stent implantation. Since coronary
37
38 angiography provides only a luminal image, stent size calculation is difficult, especially
39
40 in long dissections. IVUS in this setting allows accurate calculation of vessel size. In
41
42 coronary dissection there is a risk of inadvertently wiring the false lumen. IVUS assures
43
44 that the guide-wire is placed in the true lumen, yielding safe stent implantation. In the
45
46 present case, IVUS helped us to localize the ~~door~~ entrance towards the false lumen
47
48 and to understand the mechanism of recurrence of angina. Distal LAD artery occlusion
49
50 was seen at the apical level in the second coronary angiogram. IVUS suggested that
51
52 the ~~door~~ entrance was a few millimeters beyond the origin of the first diagonal artery
53
54 and that occlusion of the distal LAD may have been responsible for the ~~was ascribed to~~
55
56 sudden re-bleeding into the false lumen on the third day, causing downwards
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2 expansion of the hematoma. This possibility indicates that close surveillance is needed
3
4 in patients receiving conservative treatment. ~~A multidetector CT angiography or~~
5
6 ~~coronary angiogram is recommended to evaluate the healing or progression of the~~
7
8 ~~dissection at follow-up.~~

9
10 In summary, the present case describes the failure of conservative therapy in a woman
11
12 with spontaneous coronary dissection, highlighting the relevance of IVUS in diagnosis,
13
14 and illustrating the good results with stent implantation.
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For Peer Review

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LEGENDS

Figure 1. A: The coronary angiogram showed a diffuse luminal narrowing of the anterior descending coronary artery from close to the first diagonal to the apical segment due to spontaneous coronary dissection (arrows). **B:** Coronary angiogram after stenting.

Figure 2. Above: Angiogram showing occlusion of the apical segment of the anterior descending coronary artery. Numbered cross-sectional intravascular ultrasound images and the corresponding transversal segment in the coronary angiogram of the anterior left descending coronary artery are shown. **Below:** Four ultrasound images are shown. Slice 1 shows an ultrasound image with a small atherosclerotic plaque in the arterial wall and the origin of the first diagonal branch. No image of dissection is seen. Slice 2. Note the hypodense images in the slice 2 suggestive of recent bleeding. Slice 3. Image of an organized hematoma encircling almost the entire circumference of the vessel. Slice 4. Image of an organized hematoma with internal hyperdense areas of unclotted blood. are seen in the slices 3 and 4.