Spontaneous coronary artery dissection: complete angiographic resolution without stenting

A case of spontaneous coronary artery dissection in a 49-year-old woman is presented. She did not present the classical cardiovascular risk factors. Etiology and treatment are discussed. She underwent primary percutaneous coronary intervention of the left anterior descending artery with no stenting and had complete angiographic resolution.

Keywords: Aneurysm, dissecting/diagnosis; Coronary aneurysm/diagnosis; Myocardial infarction; Case reports [Publication type]

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome affecting young patients especially females with no classical cardiovascular risk factors\(^1\).\(^2\). It is a clinically relevant syndrome with a reported short-term mortality over 40\%\(^3\).

The exact etiology remains unknown. However, medial eosinophilic angiitis\(^4\), pregnancy-induced degeneration of collagen\(^4\), antiphospholipid antibodies, and deficiency of enzymes related to metabolism of the extracellular matrix\(^5\) are all possible underlying mechanisms.

Usually SCAD involves a large extension of the epicardial vessel during the acute phase. As a result, percutaneous treatment may be challenging because the risk of wiring the false lumen and the need of a long segment stenting\(^6\) increases the risk of the index procedure and the likelihood of late restenosis.

On the other hand, the spontaneous healing of SCAD has been well documented\(^7\)-\(^8\) even in cases with multivessel spontaneous coronary dissection\(^9\), which makes the medical treatment an important option for these patients. However, SCAD in the setting of an ST-elevation myocardial infarction (STEMI), persisting chest pain or hemodynamic compromise should encourage prompt re-establishment of coronary blood flow.

We report a young female presenting with anterior ST-segment elevation myocardial infarction due to SCAD, who underwent primary percutaneous coronary intervention (PCI) of the left anterior descending artery (LAD) without stenting and its complete angiographic resolution.

Case Report

A 49-year-old African-American woman, resident in the US and temporarily living in São Paulo, Brazil, presented to the emergency department with...
intense chest pain radiating to the left arm and lasting about 60 minutes. On examination, she was a healthy woman, 163 cm tall and weighing 60 kg with no reference to established risk factors for atherosclerosis, prior cardiac disease, trauma, collagen-vascular disease or use of contraceptive and recreational drugs. Her blood pressure was 139x85 mm Hg and heart rate was 60 beats/min.

The ECG showed a regular sinus rhythm and 3mm ST segment elevation in leads V2 to V6, D2, D3 and AVF (Figure 1). Cardiac markers were elevated with peak CK-MB 185 ng/ml (normal < 4) and troponin I 82 ng/ml (normal < 0.4). Full blood examinations were normal and are depicted in Table 1.

<table>
<thead>
<tr>
<th>Laboratory results and cardiac markers during hospital stay.</th>
<th>April 10, 2006</th>
<th>April 12, 2006</th>
<th>April 14, 2006</th>
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<tbody>
<tr>
<td>Hematocrit, %</td>
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<td>6.7</td>
</tr>
<tr>
<td>Hemoglobin, g/l</td>
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<td></td>
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<tr>
<td>Leukocyte count, mm$^3$</td>
<td>11900</td>
<td>4</td>
<td></td>
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<tr>
<td>Platelet count, mm$^3$</td>
<td>315000</td>
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<td>Total cholesterol, mg/dl</td>
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<tr>
<td>HDL-cholesterol, mg/dl</td>
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<td>105</td>
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<tr>
<td>LDL-cholesterol, mg/dl</td>
<td>94</td>
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<tr>
<td>Triglycerides, mg/dl</td>
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<td>162</td>
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</tr>
<tr>
<td>CK-MB, ng/ml</td>
<td>185</td>
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<td></td>
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<tr>
<td>Troponin I, ng/ml</td>
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<tr>
<td>BNP, pg/ml</td>
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<tr>
<td>ESR, mm/h</td>
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<tr>
<td>TSH, mu/ml</td>
<td>4.3</td>
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<tr>
<td>T4, ng/dl</td>
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<td>0.80</td>
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<tr>
<td>Glycemia, mg/dl</td>
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<td>105</td>
<td></td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td>0.9</td>
<td>0.9</td>
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</tr>
</tbody>
</table>

Table 1: Laboratory results and cardiac markers during hospital stay.

BNP = Brain natriuretic peptide; ESR = erythrocyte sedimentation rate; TSH = Thyroid-stimulating hormone; T4 = Free Thyroxine

Aspirin, oxygen, beta-receptor blocker and intravenous nitrate were initially given. She was referred to the catheterization laboratory as an emergency case. Coronary angiography demonstrated a long dissection of the LAD compromising blood flow (TIMI 1) (Figure 2A).

There were no findings of atherosclerosis elsewhere.

Since the distal flow of the LAD was severely impaired, associated with ongoing chest pain and ST segment elevation, an interventional strategy was planned. Heparin (80 IU/kg) and abciximab were given.

The lesion was crossed with a 0.014” floppy wire, supposedly by the true lumen, producing an immediate improvement of flow, with chest pain resolution (Figure 2B and 2C). After re-establishment of coronary blood flow, the wire was removed and the patient was maintained under conservative medical treatment. No balloons were inflated or stents placed.

After three days, a multidetector CT angiography (64-slice Toshiba scanner) was undertaken to evaluate the LAD dissection. There were still clear signs of luminal compression of the middle LAD but with distal preserved blood flow (Figure 3). The calcium score was zero.

The patient remained with no symptoms during the hospital stay, but considering the large myocardial area at risk and the signs of luminal involvement in the multidetector CT angiography, we kept the patient under medical treatment with scheduled angiographic restudy. The patient was discharged on aspirin, beta receptor-blocker, statin and clopidogrel.

After five months, we performed a new coronary angiogram that showed a normal LAD with complete resolution of the dissection (Figure 4).
Figure 2. Coronary angiographic images of the left anterior descending artery (LAD) demonstrating the long segment dissection. A – LAD with compromised distal flow (arrow). B – Wire crossing the true lumen distally (arrow). C – Wire removed and distal flow restored.

Figure 3. Multidetector coronary angiogram demonstrates luminal compression due to intramural hematoma (arrows) extending from middle to the distal portion of left anterior descending artery.
DISCUSSION

Spontaneous coronary artery dissection is a rare but increasingly recognized non-atherothrombotic cause of acute coronary events. The diagnosis of SCAD can be easily missed due to: a) sudden death clinical presentation in approximately 50% of cases\(^{(3)}\); b) possible normal or difficult interpretation of coronary angiography\(^{(10)}\); c) occurrence in healthy people with no cardiovascular risk factors.

The first description was made by Pretty, in 1931, in a post-mortem examination\(^{(11)}\). The precise etiology remains unknown. However, SCAD has been documented more often in young women in the context of use of contraceptives or during the peripartum period\(^{(12)}\). The association of high levels of estrogen, which affect collagen metabolism and the concurrent increased ‘shear stress’ is the most accepted hypothesis for the occurrence of SCAD in the third trimester of pregnancy. Also SCAD has been associated to drug abuse\(^{(13)}\), sleep deprivation\(^{(14)}\), antiphospholipid and anticardiolipin antibodies\(^{(15)}\), deficiency of enzymes, such as lysyl oxidase (LOX), which are necessary to the metabolism of extracellular matrix\(^{(5)}\), as well as to collagen vascular disease\(^{(16)}\). The possible pathogenesis is related to coronary artery disruption with hemorrhage and formation of hematoma that progressively compress the true lumen of the vessel leading to the acute coronary syndromes.

Currently, with the widespread use of coronary angiogram in the setting of acute coronary syndromes the diagnosis is made more frequently. Moreover, with new image modalities, such as intravascular ultrasound and multidetector CT angiography, it is expected that new insights into the complex pathophysiology of SCAD will come out. The optimal management remains unknown and should be individualized depending on the clinical presentation, anatomy of dissection, involvement of one or multiple vessels, and coronary blood flow. Our patient presented with STEMI with a long mid- and distal LAD segment dissection compromising coronary blood flow.

Based on angiographic findings of the LAD, an alternative diagnosis, such as isolated coronary spasm, was not considered because the acute attack of coronary arterial spasm can be promptly relieved by the administration of nitroglycerin. Whether coronary spasm occurs in association with spontaneous coronary artery dissection is still unknown. Likewise the diagnosis of chronic total occlusion (CTO) is frequently associated with diffuse coronary artery disease, coronary calcification, and chronic symptoms and still represents a challenge to percutaneous treatment. In this case, the acute presentation, normal angiographic appearance of other coronary arteries, calcium score (zero) and the undemanding wire crossing through LAD lesion helps to exclude CTO as the primary diagnosis.

After crossing the affected segment with a floppy wire, the coronary blood flow improved. The most probable explanation for such improvement is related to the mechanical action of the wire, widening the true lumen of the vessel and improving the distal blood flow. The role of concomitant pharmacological treatment with
antiplatelet and anticoagulant agents in this context would be to improve the resolution of hematoma and prevent thrombosis, favoring healing of the coronary artery dissection.

The experience of previous cases and the close contact with interventional neuroradiologists, witnessing spontaneous resolutions of vertebral artery dissections, made us decide to keep the patient under conservative treatment in this situation, avoiding to stent a very long coronary segment that could increase the risk of restenosis and stent thrombosis. Nevertheless, some authors reported clinical success after PCI in the setting of SCAD with bare stents and drug-eluting stents.

Given the large myocardial ischemic area supplied by the LAD, we decided to perform a multidetector CT angiography to evaluate healing or progression of the SCAD. It demonstrated a reduction of the true lumen of LAD but with distal preserved coronary blood flow. The control coronary angiography showed a complete resolution of SCAD and an improved left ventricular systolic function, confirming that conservative medical treatment was an excellent therapeutic strategy and possibly the most indicated. In cases where normal flow is already present at the initial angiogram, we suggest to do no further intervention. On the other hand, in cases with compromised flow, passing the wire through the true lumen may be sufficient to reestablish coronary blood flow.

CONCLUSION
We reported a successful and unusual percutaneous coronary treatment for myocardial infarction with ST segment elevation due to spontaneous coronary artery dissection with complete angiographic healing and left ventricular systolic function recovery after five months. Furthermore, the multidetector CT angiography can be a useful tool to follow up these patients, hence avoiding repeated invasive imaging.

REFERENCES