Multivessel spontaneous coronary dissection presenting as acute anterior STEMI

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Abstract

This paper reports the case of a 38-year-old woman who presented with an acute anterior ST-elevation MI secondary to multivessel spontaneous coronary dissections involving the left main, anterior descending, first diagonal and obtuse marginal branch. Stenting was performed to resolve the coronary occlusions.

A young woman, active smoker and hypertensive but without atherosclerotic risk factors, estrogen therapy or autoimmune disease, entered the ER for prolonged heavy chest pain with a duration of 1 hour, associated with mild shortness of breath, and unrelated to exercise or emotional stress.

Clinical history included percutaneous treatment of mid-cerebral artery aneurysm 10 years before and residual small anterior aneurysm under clinical and diagnostic follow-up.

On admission, Electrocardiogram showed sinus rhythm and 6-mm ST-elevation in anterior leads. On physical examination, the patient was in good and stable haemodynamic conditions: heart rate 80 bpm, blood pressure 110/70 mmHg and oxygen saturation 99%. No signs of heart failure were present.

The patient was immediately given 500 mg of Aspirin, 600 mg of Clopidogrel, 5000 UI of heparin i.v. and entered the cath-lab for an emergent coronary angiogram. The angiogram showed total occlusion of the left anterior descending artery (LAD) at the ostial segment and a dual lumen involving the left main (LM) with a huge vessel hematoma compressing the lumen, as confirmed by a subsequent intravascular ultrasound (IVUS) evaluation (Opticross 40 Mhz, Boston Scientific Corp, Natick, Massachusetts) **(Figure 1).**

A highly supportive 0.014" hydrophilic guide wire (Sion Blue, Asahi Intecc Co, Ltd) was passed to the anterior descending artery obtaining only a suboptimal flow restoration on a first diagonal branch (D1 or parallel LAD) due to type F dissection according to NHLBI Classification **(Figure2).** Therefore two drug-eluting stents (Xience Sierra 48 mm, 3.0 + 18 mm, 2.5; Abbott Vascular, Santa Clara, CA) were deployed on proximal LAD-D1, followed by LM stenting (Xience Sierra 18 mm, 4.0) in order to fix the severe lumen dissection and avoid retrograde false lumen propagation on left circumflex (LCx).

However, after LM stenting and LCx ostium postdilation, acute obtuse marginal (OM) branch occlusion occurred due to double lumen dissection, thus requiring wiring and spot stenting (Xience Sierra 18 mm, 2.5) **(Figure 3).** The IVUS demonstrated optimal stent struts expansion and apposition to the vessel wall together with a huge half-circumferential hematoma extending deeply into the media of distal LM and LAD. In order to obtain a full blood flow restoration, a high-support wire was advanced through the occlusive dissection

allowing for a good vessel visualization from middle to apex-LAD segments and an additional stent (Xience Sierra 23 mm, 2.5) implanted to avoid abrupt mid-LAD occlusion TIMI 3 flow was restored without residual stenosis except for a residual not-flow limiting dissection at distal portion of LAD **(Figure 4).** No major arrhythmias occurred during the procedure and haemodynamic stability without any drug or mechanical support was maintained over the PCI time duration and subsequently. After PCI, the patient was transferred to Coronary Care Unit still symptomatic for chest pain lasting few hours ahead.

Cardiac high-sensitivity Troponin peak reached 140900 ng/ml (reference range < 16 ng/ml) . A transthoracic echo showed normal left ventricle volumes but akinesis of septal-mid anterior and anterolateral wall segments conditioning a severe systolic function depression (LVEF 32%).

The patient remained asymptomatic and was discharged on dual antiplatelet therapy, beta-blocker, ACE-inhibitor, diuretics and statin.

A pre-discharge thorough vascular screening was conducted on carotid and vertebral arteries, renal arteries, aorta and lower limbs in order to exclude a possible concomitant fibromuscular dysplasia. Additionally, the Echocardiogram performed 7-days after acute onset and pre-discharge showed a significant improvement in global systolic left ventricular function (LVEF 42%).

Spontaneous coronary artery dissection (SCAD) is an infrequent cause of non-traumatic and non-iatrogenic tearing of the vessel wall but has emerged as an important cause of acute coronary syndrome (ACS) in patients without atherosclerotic risk factors. Its prevalence ranges between 0.2 and 4% of all ACS, and 8.7 to 35% among women younger than 50 years of age[1], oftenly detected due to a more extensive use of intracoronary imaging. SCAD still remains a deadly threat cardiovascular condition with a mortality rate of 4.2% [2,3]. Multivessel SCAD is unusual and has been described in a minority ($\sim 10\%$) of cases[4]. A recent study from the Mayo Clinic suggested that patients with intramural hematoma may develop a higher risk for progression and events compared to patients with classical angiographic dissections[5]. Spontaneous coronary dissections may be associated, in female, with a systemic immunological vascular disease (i.e. connective, inflammatory and autoimmune disorders), estrogen containing medications and stress factors [6].

In this case, well established depressive and anxiety disorders reactive to separation may have played a substantial role in the acute coronary syndrome onset together with an undiagnosed genetic predisposing setting. The choice for a prompt invasive approach to coronary revascularization was mandatory due to the patient's clinical status and the presence of acute coronary occlusions with left main stem involvement, and ongoing ischemia.

LEARNING OBJECTIVES:

1. Spontaneous coronary artery dissection (SCAD) is an under-recognized cause of acute coronary syndrome in women presenting with myocardial infarction without traditional cardiovascular risk factors
2. Intravascular imaging may play a substantial role in detection of SCAD
3. The use of medical therapy for its treatment is still empirical while the choice to perform a revascularization depends on patient's clinical setting and on angiographic coronary aspect.

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