Left Main Acute Myocardial Infarction Associated with Limited Aortic Dissection-Pandora's Box

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Abstract

Acute aortic dissection is a relative rare disease which can sometimes mimic acute myocardial infarction, usually inferior, secondary to right coronary artery involvement. Accurate rapid diagnosis is mandatory for successful treatment and usually implies cardiac surgery with the correction of arterial wall.

We present a case of spontaneous limited aortic dissection of the left Valsalva sinus, complicated by non-ST elevation MI (non-STEMI) presented to the emergency room as cardiogenic shock and successfully treated by emergent angioplasty of the left main, therapy which proved lifesaving as a bridge to surgery.

The case highlights that even in front of a well-defined clinical, ECG and biological presentation suggestive for acute MI, one should always be mindful for the differential diagnosis of acute aortic dissection.

The interventional treatment in this critical situation was effective due to the pathological peculiarity of the dissection, which was strictly focal and limited and could be stabilized by left main stenting.

This case also underlines that treatment should always be adapted to the patient's disease, and that sometimes therapies with an absolute contraindication may be a lifesaving solution in a specific context.

Keywords: Left main acute myocardial infarction; Cardiogenic shock; Aortic dissection; Sinus of Valsalva

Case Report

A 68 year-old hypertensive and dyslipidemic female, with known permanent Atrial Fibrillation (AF) and no treatment, presented to the territorial hospital for typical retrosternal chest pain radiated to the upper limbs, accompanied by dyspnea, orthopnea and confusion, symptoms started within 24 hours before admission. She was diagnosed with acute coronary syndrome, received anti-thrombotic therapy (Dual Anti-Platelet Therapy (DAPT) and Unfractionated Heparin (UH)) and anti-anginal medication (IV Nitroglycerin), and was addressed to our hospital for emergent coronary angiography.

On admission, the patient's clinical condition had significantly deteriorated, with signs of acute pulmonary edema and cardiogenic shock criteria. The ECG (Figure 1) showed AF with rapid ventricular response, narrow QRS with diffuse ST depression (maximum 1 mm) and ST elevation in a VR, suggestive for possible left main (LM) injury or LM equivalency.

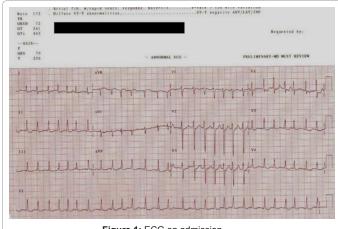


Figure 1: ECG on admission.

The laboratory work out showed the presence of a biological inflammatory syndrome and high Troponin level, confirming the diagnosis of acute MI.

The clinical, ECG and biological evaluation confirmed the diagnosis of very high risk non-STEMI, with cardiogenic shock and acute pulmonary edema. The calculated GRACE risk score [1] was 259 for in-hospital death (mortality 50%) and CRUSADE score [2] was 71 (19.5% risk of bleeding). She was referred for emergent angiographic evaluation within 2 hours from admission [3], under pharmacological

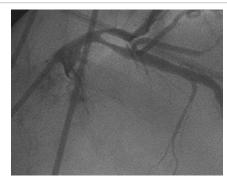


Figure 2: RAO cranial wiew: 1. LM stenosis of 50%, 2. LAD ostium stenosis of 80%, 3. CxA ostium stenosis of 80%.

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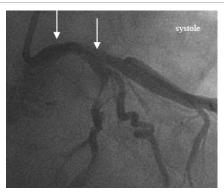


Figure 3: RAO caudal view: the aspect of repletion-depletion in the LM and the ostium of LAD during the cardiac cycle.

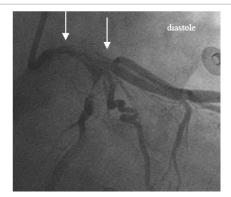


Figure 4: RAO caudal view: the aspect of repletion-depletion in the LM and the ostium of LAD during the cardiac cycle.

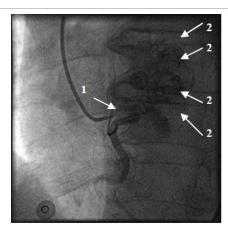


Figure 5: LAO 30°: 1.RCA with atypical origin 2 dilatation of the left Valsalva sinus.

hemodynamic support and non invasive ventilation (CPAP).

The diagnostic angiography revealed significant LM stenosis (Figure 2), with involvement of the Left Anterior Descending artery (LAD) and Circumflex Artery (CxA) ostia, both with critical 80% stenosis. The fusiform shapes of both ostia stenosis and a movement of repletion-depletion in the LM during the cardiac cycle (Figures 3 and 4) suggested the presence of a dissection fold at this level. The Right Coronary Artery (RCA) showed atypical anterior origin, but without associated pathology. Non-selective injection in the aorta during RCA examination revealed a dilated left Valsalva sinus (Figure 5).

The angiography concluded to the presence of significant LM stenosis with ostia involvements of both LAD and CxA, while raising the suspicion of spontaneous dissection of the LM with distal propagation of the fold to the origins of LAD and CxA.

Although the suspected pathology would have required further investigations, given the critical state of the patient, with signs of evolving AMI complicated with severe pump dysfunction and cardiogenic shock, in view of the large mass of myocardium at risk, it was decided to perform angioplasty of the LM and the origins of LAD and CxA. PCI was performed using two bear metal stents (LMLAD/CxA), with good angiographic result (TIMI 3 distal flow, blush 3) (Figure 6).

The patient's evolution was favorable, with acute heart failure and cardiogenic shock remission within the first 24 hours following the procedure, stabilizing to NYHA class II HF, without angina.

The Trans Thoracic Echocardiography (TTE) showed a dilated ascending aorta of 45 mm, with a small dissection fold and moderate aortic valve regurgitation, a mildly dilated Left Ventricle (LV) with segmental wall motion abnormalities consistent with LM/LAD/CxA infarction and global LVEF of 20%.

The trans-esophageal echocardiography (Figure 7) visualized an aortic dissection fold limited to the left Valsalva sinus, without extension above the sinotubular junction to the ascending aorta. The aortic root was measured at 46 mm. There was moderate aortic valve regurgitation by aortic ring dilatation and incomplete coaptation of the leaflets, without morphological damage of the valve.

The CT scan of the ascending aorta, revealed the presence of

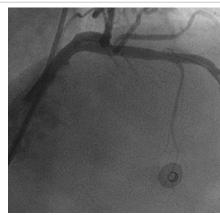


Figure 6: RAO cranial view: final result after implantation of BMSx2 I M-I AD/CxA



Figure 7: Transesophageal echocardiography: above the aortic valvular plane-patent stent in the LM and aortic fold without flow at Doppler examination.





Figure 8: CT scan: left Valsalva sinus dissection fold (left) and dilated ascending aorta (right).

an intimal fold in the left Valsalva sinus, without extension to the ascending aorta, without periaortic extravasation of contrast medium, with dilatation of the ascending aorta at 48 mm (Figure 8).

The 7 days TTE control showed the same aspect of the dissection fold and a recovery of the left ventricular systolic function with global LVEF of 40%. The final diagnosis for our patient was type II aortic dissection exclusively involving the left Valsalva sinus, with extension to the left coronary artery, complicated with acute myocardial infarction without ST elevation and cardiogenic shock, treated by PCI with implantation of two BMS resulting in normalized left coronary blood flow and stabilization of the dissection fold. The long-term treatment consisted in DAPT with Aspirin 75 mg/day and Clopidogrel 75 mg/day, cardio-selective beta-blocker (Metoprolol 50 mg/day), ACEI (Enalaprilum 5 mg/day), anti-aldosterone (Spironolactone 25 mg/day) and statin (Simvastatine 40 mg/day).

The evolution was free from cardiovascular events and the 3 month assessment showed a patient with good general condition, in NYHA class II HF, free of angina, with patent stents without restenosis to the angio follow-up. The patient is currently under heart team surveillance, with no need for surgery at 1 year of follow-up.

Discussions

Non-traumatic acute aortic dissection is a relatively rare condition with rapidly progressive evolution towards death in the absence of prompt diagnosis and treatment. The incidence of the acute aortic dissection in the general population is estimated to be 2.6-3.5/100000/ year [4,5]. Acute myocardial infarction secondary to the aortic dissection is rare, occurring in 1-2% of all cases of dissection, commonly affecting the RCA [6].

In our case, a medically neglected 68 year old hypertensive woman with permanent AF and most probably pre-existing but ignored aortic pathology, the clinical presentation of acute aortic dissection was that of a non-STEMI complicated with cardiogenic shock and acute pulmonary edema. The spontaneous dissection fold was shown by echocardiography and CT to be strictly localized to the left Valsalva sinus with left main extension but no involvement of the aorta above the sino-tubular junction. Isolated Valsalva sinus dissection is usually iatrogenic, secondary to cardiac catheterization, with only one case in the literature describing a similar to our case left spontaneous form [7].

The therapeutical decision to perform PCI on the LM-LAD and CxA turned out to be ideal as it resulted in return to normal blood flow in the left coronary artery and stabilization of the dissection fold, with no need for surgical intervention at one year follow-up. It also lead to

a dramatic decrease of the Euro SCORE II predicted mortality risk for cardiac surgery, from 80.36% in the acute setting to 8.46% at one month [8].

The interventional solution proved beneficial and, to our knowledge, it is for the first time described in a case of spontaneous Valsalva sinus dissection. In this particular case the life-threatening condition was the acute ischemia of the LM, so treating it proved to be the solution for the survival of our patient.

The left ventricular systolic dysfunction improvement was consistent with an ischemic etiology and not related to the aortic valve regurgitation, whose degree remained constant throughout the evolution of our patient. This suggests a pre-existing aortic valve disease, concordant with the ascending aorta dilation. The co-existence of aortic dissection, PCI with BMS on the LM and permanent AF with a CHADS2-VASc risk score of 4 for thrombo-embolic events [9] represents an important issue regarding the optimal anti-thrombotic treatment.

Conclusions

This case is a reminder that aortic dissection can mimic myocardial infarction and should always be considered when dealing with differential diagnostic of acute coronary syndromes.

Spontaneous limited to Valsalva sinus dissection with coronary involvement is a very rare entity, difficult to diagnose. In this specific setting, coronary angioplasty can be considered as a therapeutical option, primarily as a bridge to surgery.

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