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EC CARDIOLOGY
Case Report

Acute Myocardial Infarction with Simultaneous ST Segment Elevation of Two Main Epicardial Vessels

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Abstract

Male, 49 years old, hypertensive patient, dyslipidemic, smoker, without previous cardiovascular history, who entered to the emergency service of our Institution, with angina pectoris of 40 minutes of evolution. He presented with evolutionary electrical changes, in sinus rhythms, with supra-elevation of the anterior ST segment and conduction disorders in the right branch with ventricular extrasystoles, Killip and Kimball A (KKA). We solved these case with simultaneous direct angioplasty (PCI) of two vessels with implantation of pharmacological stents.

Keywords: *Acute Myocardial Infarction; ST Segment; Epicardial Vessels; Killip and Kimball A (KKA)*

Introduction

Marcus DeWood in his originally article forty years ago, teaches us that acute myocardial infarction are fundamentally due to a fresh, white fibrin thrombus, that compromises the light of the vessel.

Case Report

A 49 year old patient, without prodromes and previous cardiovascular history, with an acute coronary syndrome, was presented to the emergency room in KKA. Routine medical treatment was performed (NTG, AAS, Beta blockers intravenous, clopidogrel and anticoagulation). He was sent immediately to the Cath Lab room for direct angioplasty (PCI).

Outcome

Femoral cinecoronary angiography was performed: thrombotic right coronary occlusion was observed in the third middle, with TIMI I flow; The Left Main without lesions, circumflex without lesions, and the Left Descending Anterior artery (LAD) with acute thrombotic occlusion of the proximal third and TIMI 0 flow. Ad hoc, was performed first PCI of LAD with drug-eluting stent, Medtronic 3.5 x 22 mm at 14 ATM, achieving TIMI III flow, without residual stenosis. Subsequently, PCI was performed with the under-size predilatation technique, and the implantation of two 3.5 x 33 DES Biomatrix, with overlap, carried out 14 ATMs with a 4.0 mm balloon, achieving TIMI III flow and without residual stenosis. Reperfusion is achieved in the Cath lab room, based on clinical, electrical and angiographic criteria. He was stable, without inotropics in the Coronary Unit, with isoelectric ST segment and with habitual complete secondary prevention and DAPT, being discharged from hospital at 72 hours.

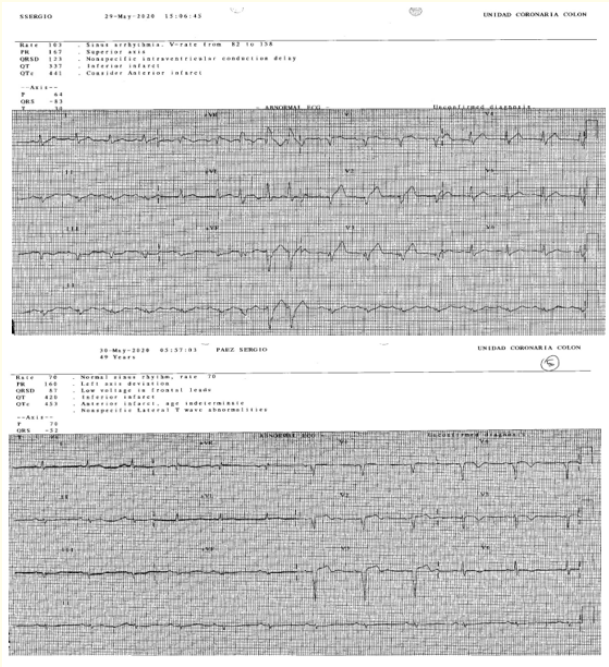

Figure 1: *Above: EKG of admission to coronary unit. Bottom: EKG post PCI.*

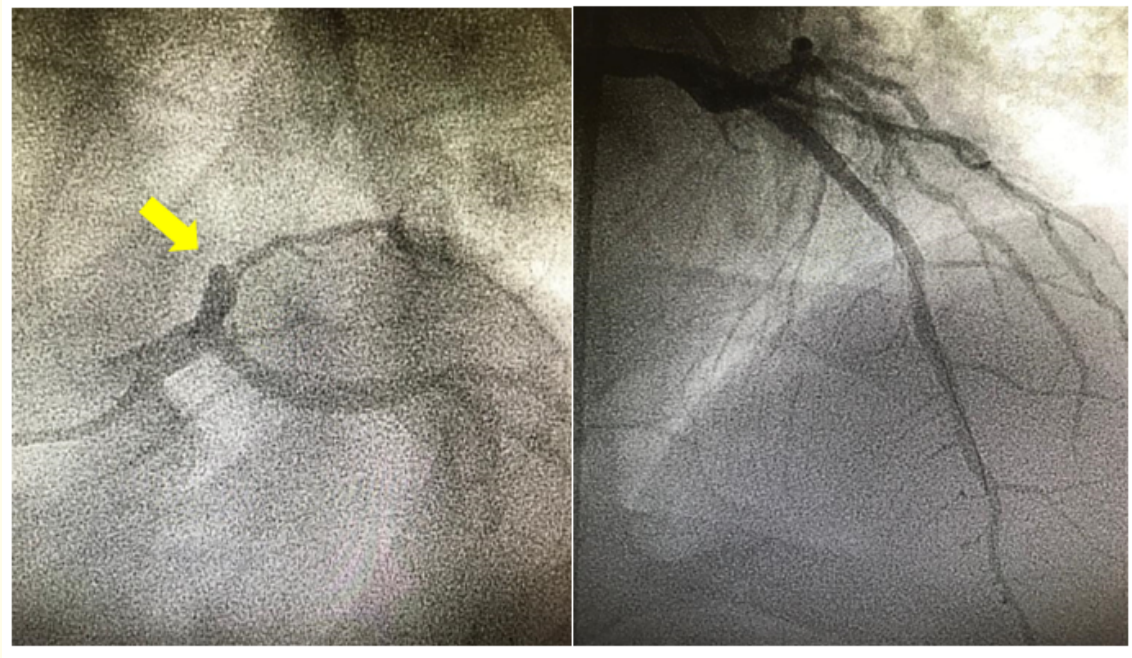

Figure 2: *Left: Acute occlusion of LAD in spider view. Right: Post PCI with stenting in ROD.*

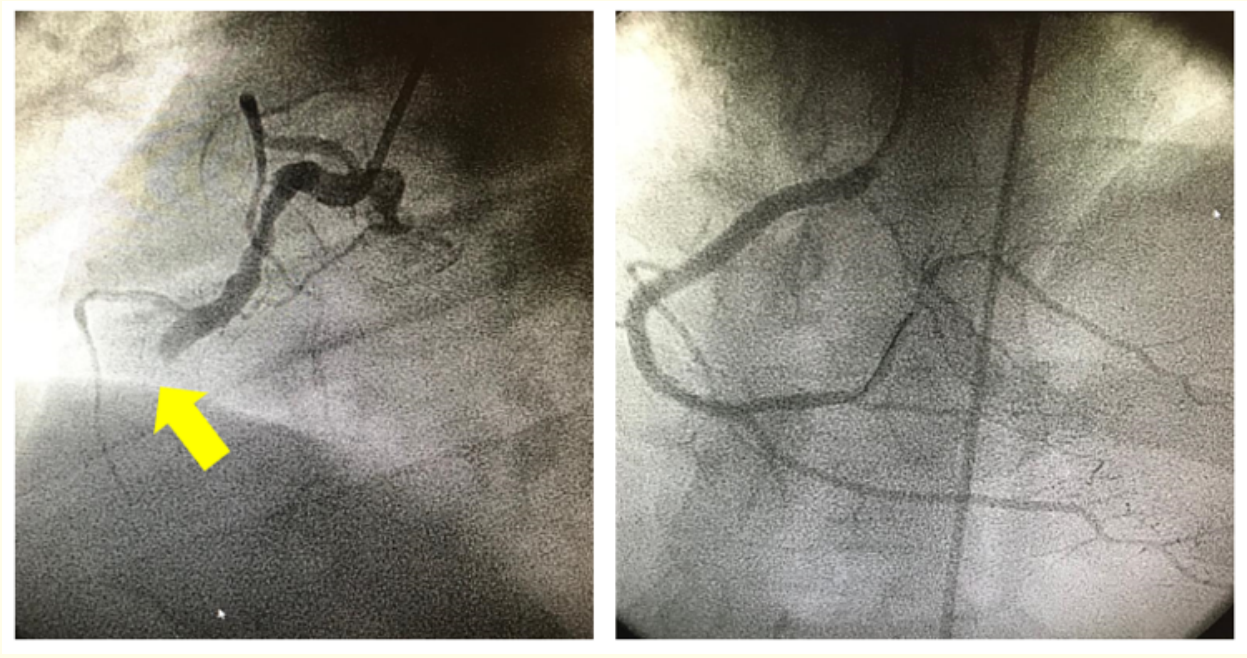

Figure 3: *Left: Acute occlusion of RCA. Right: Post PCI with two DES.*

Discussion

Marcus DeWood [1] in his originally article forty years ago, teaches us that acute myocardial infarction are fundamentally due to a fresh, white fibrin thrombus, that compromises the light of the vessel. This work was the kick off to begin the modern strategy of reperfusion. From here, different stages have been described in the revascularization of high-risk acute coronary events; as in patients with disease of more than one vessel, even the revascularization of non-guilty vessels like the current concept of prophylactic angioplasty in AMI.

When we turn to AMI in multiple vessel disease, we see that approximately 50% of patients undergoing direct PCI have diseases of more than one vessel; but is not usual to find the acute simultaneous occlusion of two large epicardials vessels with TIMI 0-I flow. This finding is reserved for a few cases of individual reporting, not exceeding 1 or 2% of the total infarcts with multiple vessel disease, because patients may present a higher incidence of sudden death and/or cardiogenic shock, due to the great myocardial mass compromised and the great electrical instability of onset and therefore not surviving the event [2].

One of the authors who has studied this phenomenon for years, was A. Maseri [3], He showed in an experimental model that there was a sort of "contagion phenomenon" and disease spread, through inflammatory factors and cytokines of the blood, carried through the great cardiac vein from the "sick vessel" to the "healthy" vessel, thus activating these "potentially vulnerable plaques" of the non-culprit territory, and thus giving rise to the simultaneous event of two vessels.

Also, as Goldestein [2] described, the concept of vulnerable unstable plaques is documented angiographically in natural history studies of patients with AMI and multivessel diseases. Even in the same patient, unstable plaques that are characterized by the inflammatory activity of the fibrous capsule and inflames these vulnerable plaques, due to a contagion effect. This could develop the expansion of intraplate contents of lipid metabolism, and with this, generate its inflammation, instability and rupture.

As we can see, there seems to be a close relationship between simultaneous acute phenomena, with the inflammation load and its potential activation and transmission by blood, focusing here on the different therapeutic possibilities for this type of patient; taking in the hypothetical terrain, the possibility of using various anti-inflammatory strategies to treat these cases, beyond the mere fact of mechanically opening the vessel to achieve reperfusion, and thus save myocardial mass and lives [4-11].

Conclusion

So many progress have been made in the knowledge and treatment of high-risk acute coronary events; but there is still a long way to walk. Different unusual stages, like the one presented here, are not well understood. We need to continue studying these patients to have a positive impact on their evolution.

Acknowledgment

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