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GIANT CORONARY ANEURYSM THROMBOSIS LEADING TO ACUTE MYOCARDIAL INFARCTION: CASE REPORT

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ABSTRACT

Coronary artery aneurysm (CAA) is defined as a localized irreversible dilation of the coronary vascular lumen with a diameter ≥ 1.5 times that of the adjacent normal coronary segment. AXP, male, 65 years old, had acute coranary syndrome and was sent to coronary unit. After 24 hours, he underwent coronary angiography that showed giant aneurysm with thrombus and total occlusion at the origin of the right coronary artery. There was an aneurysm in the left main and aneurysms in the proximal thirds of the left anterior descending artery (LAD) and left circumflex artery, and mild stenose in the LAD. This case was debated by Heart Team and was decided to refer the patient to surgery. One week latter the procedure was done and this patient was discharged on 4th after surgery. Which treatment modality (clinical, percutaneous or surgical) should be chosen for giant CAAs depends on aneurysm factors such as size, location, speed of growth and others, as well as patient factors, hospital structure, multidisciplinary team experience, etc. There are no recommendations based on guidelines for the treatment of coronary aneurysms, so the management should be individualized and according to Heart Team.

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INTRODUCTION

Coronary artery aneurysm (CAA) is defined as a localized irreversible dilation of the coronary vascular lumen with a diameter ≥ 1.5 times that of the adjacent normal coronary segment, and was first described in 1761 by Morgani. On the other hand, a diffuse dilation of a coronary artery that involves more than 50% of its extension is called ectasia. The incidence of events associated with CAA ranges from 1.5% to 5% (Elguindy MS, 2017; Sherif SA, 2017). Coronary aneurysms can be classified according to composition of the arterial layers in true, dilatations of the 3 layers of the vessels, or in pseudoaneurysm (or false aneurysm) that are dilatations of 1 or 2 layers. They are further classified according to morphology in saccular (transverse> longitudinal diameter) or fusiform (longitudinal> transverse diameter) (Elguindy MS, 2017; Sherif SA, 2017). The incidence of aneurysms ranges from 0.15% to 5.3%, being more common in men than in women.

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The right coronary artery is more commonly affected (40-70%), followed by the left anterior descending artery (15-32%), left circumflex (15 - 23%) and left main (0.1-3.5%) (Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018). The term giant coronary artery aneurysm does not have a single universal definition. Diameters greater than 20 mm, 8 mm or 4 times the adjacent reference value have been used as diagnostic criteria. The vast majority of occurs in the right coronary artery (Crawley PD, 2014). We described a case of a patient (who has given the informed consent) with acute coronary syndrome secondary to thrombosis of a giant coronary aneurysm.

CASE REPORT

AXP, male, 65 years old, married, catholic, white, lawyer, complained of retroesternal oppression radiating to the left upper limb, associated with nausea and vomiting that initially he thought to be due to digestive system and decided to take analgesics and not seek medical help. He was hypertensive taking captopril and diabetic taking insulin.

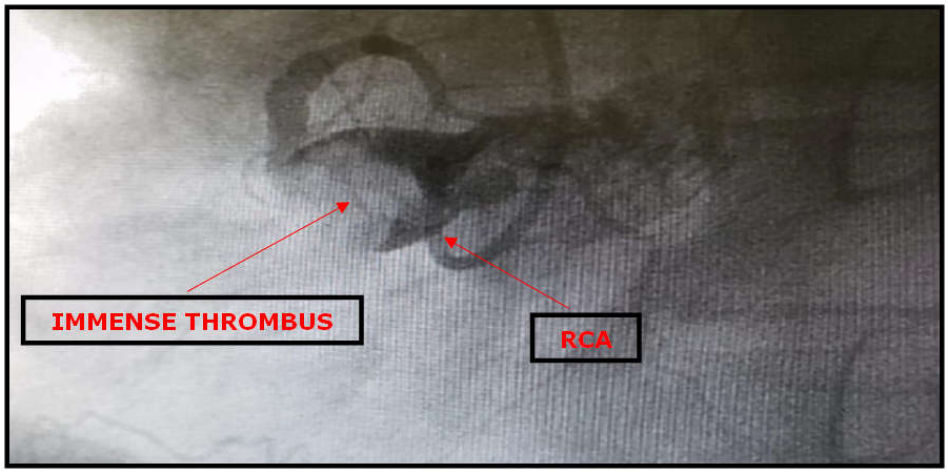

Figure 1. Giant coronary artery aneurysm with immense thrombus leading to right coronaryartery (RCA) occlusion

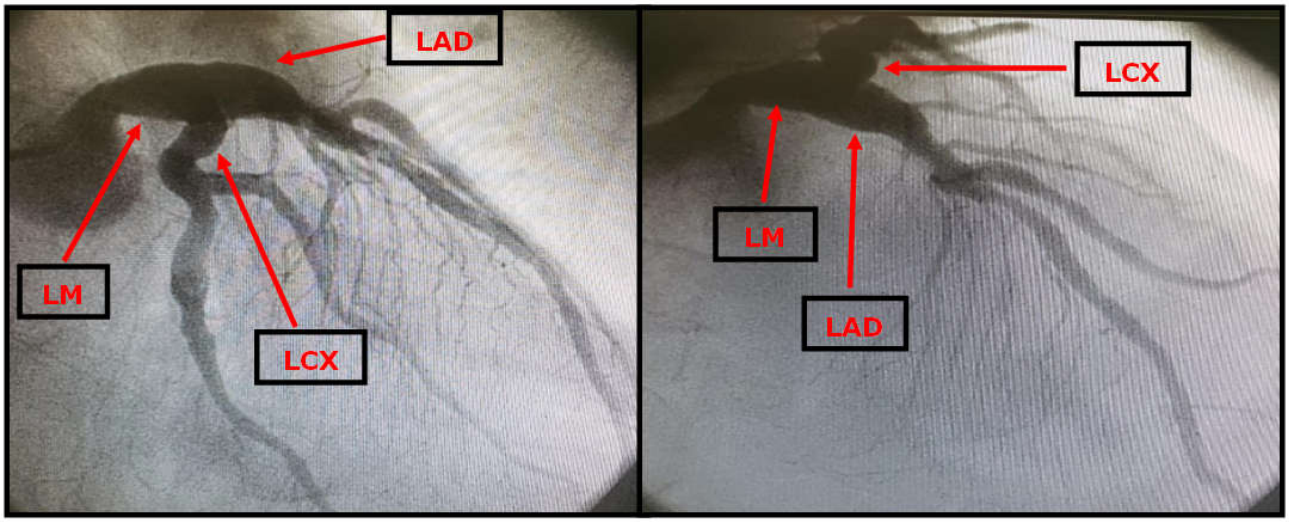

Figure 2. Leftmain (LM), left anterior descendingartery (LAD) andLeftcircumflex artery (LCX) aneurysms

He had been a smoker for 40 years. His father had suffered acute myocardial infarction at age of 42 years old. Since there was no improvement in symptoms after 1 hour and 30 minutes, he went to hospital where he underwent electrocardiogram, which had no signs of ischemia or myocardial injury, and was tretaed with aspirin, ticagrelor, beta blocker, nitrate, statin and enoxiparin. Laboratory tests were collected. Fifteen minutes after initial treatment the patient became asymptomatic, but there was elevation of troponin and was decided to send him to coronary unit. The next day he underwent coronary angiography that showed giant aneurysm (15 mm) with thrombus and total occlusion at the origin of the right coronary artery (Figure 1). There was an aneurysm of the left main (10 mm) and aneurysms in the proximal thirds of the left anterior descending artery (LAD) and left circumflex artery (LCX) , and mild stenose in the LAD (Figure 2). As the echocardiogram revealed akinetic inferior wall was decided do not perform intervention that time. This case was debated by Heart Team and was decided to refer the patient to surgery.

One week after the suspension of ticagrelor the surgery was performed. The aneurysms were ligated and the coronary arteries revascularized. The patient was discharged 4 days after surgery, taking aspirin, ticagrelor, metoprolol, atorvastatin, captopril and insulin. Six month clinical follow up revealed he was asymptomatic.

DISCUSSION

Giant coronary artery aneurysms are often diagnosed when performing coronary angiography for other reasons and have long period without symptoms, so her real prevalence is not well established, but according to available data ranges from 0.02% to 0.2%. (Elguindy MS, 2017; Crawley PD, 2014). Computed tomography, cardiac resonance angiography and echocardiography can establish the diagnosis of CAAs, but the gold standard still is the coronary angiography. If coronary intervention is planned intravascular ultrasound and the optical coherence tomography are importante and add informations because reveal a better visualization of the layers of the arteries. (Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018) The described patient had an acute myocardial infarction due to occlusive thrombosis of the aneurysm. Patients with coronary aneurysms can have angina pectoris, acute coronary syndrome, sudden death, fistulas, hemopercardium, cardiac tamponade, compression of structures, superior vena cava syndrome, etc. (Elguindy MS , 2017; Crawley PD , 2014; Boyer N, 2014). In the literature, cases of aneurysms associated with significant stenoses of the coronary arteries have been described, but in our case there was no significant stenosis. However, due to the absence of other factors associated with coronary aneurysms and the presence of

stenose, even if not significant, we believe that atherosclerosis is the cause of aneurysm formation. Atherosclerotic aneurysms are usually multipli and involve more than one coronary artery as compared with congenital, traumatic or dissecting aneurysms (Elguindy MS, 2017; Boyer N, 2014; Hada Y, 2017). It is much more common for coronary artery disease to determine lumen reduction than aneurysm formation, but it is a mistake to think that this means a reduction in coronary artery size more frequently. Indeed, in many patients, vascular remodeling with extrinsic vessel growth occurs to accommodate the atheroma and prevent lumen obstruction, and only after failure of this compensatory mechanism is there a stenosis (Schoenhagen P, 2011). At the site of the coronary artery aneurysm there may be vasculitis, inflammation with lymphocytes, plasma cells, macrophages, eosinophils, mononuclear cells, lipid deposits, cholesterol crystals, destruction of the intima and media, hyaluronization, fibrosis, calcification, intramural hemorrhage and sometimes giant cell reaction (Elguindy MS ,2017; Sherif SA, 2017; Crawley PD , 2014). It is believed that there is a destruction of the layers of the arteries leading to weakening and thinning of the arterial wall, which when exposed to stress suffers dilatation, increasing the diameter of the vessel (Elguindy MS, 2017; Sherif SA, 2017; Crawley PD, 2014). There is an increase in inflammatory cytokines, matrix metalloproteinases, enzymes (MMPs) that degrade elastin, collagen, proteoglycans, laminin and fibronectin. Besides, the inhibitory factors of matrix metalloproteinases are diminished, whereas adhesion molecules like vascular cell adhesion protein 1 (VCAM 1), Intercellular Adhesion Molecule 1 (ICAM 1) and E-selectin are increased (Elguindy MS, 2017; Sherif SA, 2017; Bajaj S, 2010). Futhermore, there is a local imbalance between production and degradation of the extracellular matrix and components of the arterial wall, with a predominance of structural loss of the vascular layers and progressive dilatation (Elguindy MS, 2017; Sherif SA, 2017).

In summary during atherosclerotic coronary aneurysms formation and progression predominate the destruction and weakening of the arterial wall, mediated by the immunoinflammatory response of patients with genetic susceptibility. In some cases the atherosclerotic mechanisms that determine stenosis and dilatation of the coronary arteries occur in the same region generating stenosis and dilations so close, sometimes continuously (Elguindy MS ,2017; Sherif SA, 2017; Singh G ,2018; Crawley PD , 2014). Slow flow and uneven surface of the aneurysm are factors that predispose to the formation of thrombi, which often undergo fragmentation and embolization leading to acute coronary syndrome (Elguindy MS, 2017; Crawley PD, 2014; Hada Y, 2017). In our case there was formation of an immense thrombus that led to complete occlusion of the aneurysm and we did not confirm embolization. Different from what happened to our patient, sometimes giant aneurysms may rupture, form fistulas, or compress structures (Elguindy MS, 2017; Sherif SA, 2017; Crawley PD, 2014). Etiologies of aneurysms are congenital or acquired. The most common are coronary artery disease, Kawasaki disease, inflammatory diseases (polyarthritis nodosa, systemic lupus erythematosus, etc.), connective tissue diseases (Marfan Syndrome, Ehlers Danlos, cystic necrosis media, etc.), infection, trauma, iatrogeny, cocaine, after percutaneous coronary intervention, coronary fistulas, Takayasu arteritis, etc. The association with dilatations of the aortic artery or other arteries is not uncommon. There is a genetic susceptibility for the formation of aneurysms (Elguindy MS, 2017; Crawley PD, 2014). Our patient had a non ST segment elevation myocardial infarction. It has been treated according to guidelines (Roffi M, 2016). Within first 24 hours he underwent coronary angiography and a giant aneurysm with occlusive thrombus was diagnosed in the right coronary artery and another in the left main. NSTEMI must be treated according to guidelines and identification of reason for that syndrome is so important (Roffi M, 2016). In this case one giant coronary artery aneurysm was the cause of the myocardial infarction. An occluded artery due to immense thrombus in a giant aneurysm represents a challenge for percutaneous intervention, especially with an estimated 24-hour occlusion time as in this case. There is no I A evidence recommendation for treatment in this case (Roffi M, 2016). Treatment of CAA thrombosis with intracoronary urokinase, intravenous heparin, oral anticoagulation, intravenous eptifibatide, aspirin and P2Y12 inibitors were reported and rate of success were variable (Boyer N, 2014). From the percutaneous point of view it would be possible to open the artery with aspiration of thrombi followed by deployment of drug eluting stent (DES) or bare metal stent (BMS) as a scaffold for deployment of a covered stent to exclude the aneurysm. In other words, aspirate thrombi, implant an DES or BMS greater than the extension of the aneurysm and then implant a covered stent into the previous implanted stent (Cotter R, 2019) Of course, this is not an easy procedure, on the contrary is a challenging with considerable risks. However, chooseing percutaneous coronary intervention there is a chance of distal embolization, reduction of flow, thrombosis and / or restenosis, rupture and mal appositiont (Boyer N, 2014; Hada Y, 2017; Cotter R, 2019). Considering the delay for reperfusion associated with akinesia (echocardiogram) and Q wave (electrocardiogram) was decided do not perform percutaneous coronary intervention. The treatment of CAD may be clinical, percutaneous or surgical. There are no randomized clinical trials on the subject that allow a recommendation based on strong scientific evidence, and the treatment should be individualized and based on observational studies or reports of cases or series of patients (Elguindy MS ,2017; Sherif SA, 2017; Singh G ,2018; Crawley PD , 2014; Boyer N, 2014). Clinical treatment is directed to the cause of the aneurysm, and in addition, single or double antiplatelet therapy and in some cases the use of anticoagulants have been recommended to avoid thrombus or embolization. In the case of CAD, the pharmacological and non-pharmacological treatment of secondary prevention are recommended. The possible link between inflammatory cytokines and MMP with CAAs may also indicate a role for statins and inhibition of teh renin angiotensin system (Elguindy MS ,2017; Sherif SA, 2017).

Percutaneous intervention has been performed through the implantation of covered stents, BMS, DES and coils. Although there is a fear of increased chance of thrombosis, restenosis and compromise of side branches with the implantation of covered stents, this device has been the most used. (Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018; Crawley PD, 2014; Boyer N, 2014; Hada Y, 2017). Surgical treatment through ligation or excision of the aneurysm and myocardial revascularization has been shown to be an effective and safe treatment ((Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018). Which treatment modality should be chosen for CAAs depends on aneurysm factors such as size, location, speed of growth and others, as well as patient factors, hospital structure, multidisciplinary team experience, etc (Elguindy MS, 2017;

Sherif SA, 2017; Singh G, 2018; Crawley PD, 2014; Boyer N, 2014; Hada Y, 2017; Cotter R, 2019). Giant coronary aneurysms have been treated surgically. When the aneurysm is greater than 10 mm, symptomatic or with rapid growth velocity or in locations at high risk of events, surgery is strongly recommended ((Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018; Crawley PD, 2014; Cotter R, 2019). Our patient had a giant aneurysm in the left main and had suffered an acute myocardial infarction due to occlusive thrombus in a giant aneurysm of the right coronary artery. Therefore, a new aneurysm thrombosis, this time in the left main would be catastrophic since it would be associated with a very high chance of death. So, because of theses considerations was decided for surgery, which is in accordance with the recommendation that aneurysms > 10 mm in the left main should be operated (Crawley PD, 2014).

Aneurysms of coronary arteries are rare and there are diferente etiologies, but the most common is atherosclerosis. Aneurysms can be asymptomatic or determine angina, myocardial infarction, hemopedicardium, fistulas, cardiac tamponade, etc. The treatment of giant aneurysms is a challenge, especially when they are cause of acute coronary syndromes. If primary reperfusion is not the goal, such aneurysms are treated surgically in most cases. In the scenario of primary reperfusion the technique described in this article (aspiration, stent as an scaffold and covered stent) may be an option but represents a challenge. There are no recommendations based on guidelines for the treatment of coronary aneurysms, so the management should be individualized and according to Heart Team (Elguindy MS, 2017; Sherif SA, 2017; Singh G, 2018; Crawley PD, 2014; Boyer N, 2014; Hada Y, 2017; Cotter R, 2019).

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