

# Coronary aneurysm & Stenotic lesion



Dr. Gerardo Nau ICBA Instituto Cardiovascular de Buenos Aires

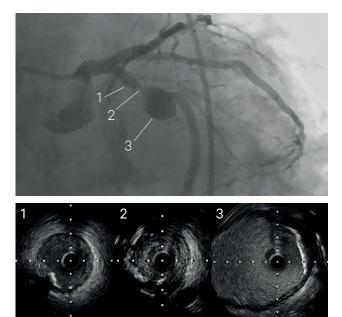
# Patient history

A 59 year old male patient with a history of hypertension and chronic renal failure, presented at the emergency department due to chest pain lasting 30 minutes. An ECG was performed in triage, T-wave inversion V4 to V6 were observed. The patient was admitted, initiating medical therapy with 300 mg of acetyl salicylic acid, low molecular weight heparin, intravenous vasodilators, satins and beta-blocker. After this first approach, he evolved asymptomatically, without hemodynamic alterations.

## Initial situation

The laboratory result showed a positive ultrasensitive troponin of 126 ng/l. The condition was interpreted as non ST elevation myocardial infarction. Ticagrelor loading dose was administered and a coronary angiography performed on the same day.

After establishing access through the femoral artery with 6 Fr sheath, we performed a diagnostic angiography, showing a chronic occlusion at the mid-level of the right coronary artery. Severe lesion on the diagonal branch without compromising the bifurcation. The proximal circumflex artery showed a severe lesion with a stenosis of nearly 90 % and a subsequent saccular aneurysmal dilation. From inside this latter, two small branches appear irrigating the lateral region without further significant lesions.



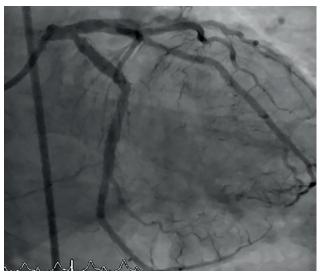
# Procedural course

We continued with coronary angioplasty, placing a XB 3.5 guide catheter. The diagonal branch was treated first. We implanted a direct drug eluting stent, without ostium involvement.

Afterwards we advanced a 0.014" Balanced Middle Weight (BMW) guide wire through the circumflex coronary (CX) artery. With a  $2.5 \times 12$  mm balloon we pre-dilated the stenotic lesion at 16 atm. After this, IVUS was done in order to assure the pre-, intra- and post-aneurysm diameters and length. It is of vital importance to recognize the vessel diameters, since the covered stents require the complete apposition to avoid leaks and at the same time avoid under-sizing, since they present a higher risk of thrombosis.

Before implanting the covered stent, we prefer to dilate the lesion with a high pressure balloon 3.0 x 12 mm at 22 atm due to heavy calcification.

To cover the stenotic lesion and aneurysm we placed a 3.5  $\times$  24 mm Bentley BeGraft coronary covered stent at 14 atm.



Contrast stentboost and IVUS showed a nice stent adaptation and freedom from leakage. Nonetheless, signs of stent under-expansion appeared, and we decided to post-dilate with high pressure balloon  $3.5 \times 15$  mm. It is suggested not to oversize covered ePTFE stents due to the risk of material damage.



## Comments

The diagnosis of coronary artery aneurysm (CAA) is described as a condition where the artery is 1.5 times larger than the adjacent arterial segment. The incidence of coronary aneurysms varies from 0.3 % to 5.3 %. (a) They can be saccular like in our presentation or fusiform. Up to 70 % of aneurysms appear within the right coronary artery whereby ca. 23 % occur in the circumflex artery.

The physio-pathological mechanism continues to be controversial, being half in adults associated with atherosclerotic disease. Chronic inflammation results in the weakening of the arterial wall, destruction of the muscular elastic layer, fibrosis and calcification. Moreover, the vast majority of aneurysms reveal an association with coronary stenosis as exemplified in our case. (c,d,e)

In most cases CAA evolve asymptomatically. In the case of manifesting symptoms, they are in greater proportion ischemic due to the formation of internal thrombus and embolization and/or alteration of the blood flow. Even more acute coronary syndromes can be caused due to their association with stenotic lesions. In our case it is showing different patterns of the same disease.

There is no consensus on the proper management of CAAs, since the natural history of the disease is based on multiple factors which make it necessary to customize the treatment according to the patient. For that reason, before taking an interventional decision, we must consider the clinical presentation, possible etiology (infectious, atherosclerosis, connective tissue disorders, vasculitis, etc.), aneurysm size and expansion on follow up.

Today percutaneous treatment is a less invasive option than open surgery, however scarce data is obtained. One of the largest studies retrospectively compared outcomes in a series of patients treated with either surgery (n = 18) or covered stents (n = 24). Patients treated with covered stents tended to be older (60.5 vs. 47.7 years old) and to have smaller aneurysms (9.8 vs. 35.1 mm). No deaths were reported in either group. Only 5 of the 24 patients who received stents were found to have restenosis on follow-up angiography. (f)

Due to the new technological developments such as the Bentley BeGraft coronary a much better trackability and long term safety has been obtained. These two missing qualities diminished the percutaneous choice in the past.

Percutaneous treatment and the decision for exclusion should be considered in anatomies that do not exclude important branches. Also, we strongly suggest angiography-supported images in order to reduce the risks of thrombosis and restenosis in follow-up. The use of IVUS is extremely helpful. Since suitable lengths and diameters are available it is becoming more and more the gold standard for the evaluation of the vessel wall and aneurysmal diameters. IVUS variables are described as anatomic risk factors for rupture, encourage us to take an invasive stand.

In our case the percutaneous treatment of the aneurysm was due to main findings in IVUS and the unstable stenotic disease. Our knowledge of coronary artery aneurysms is limited, and management is still a challenge. New tools to recognize high risk patterns and devices to deal with these complex anatomies make us a great progress. Through its excellent performance the BeGraft coronary is a very helpful device which we are using regularly for such challenging cases.

#### References

- Hartnell GG, Parnell BM, Pridie RB. Coronary artery ectasia: its prevalence and clinical significance in 4993 patients. Br Heart J. 1985;54:392–395.
- Villines TC, Avedissian LS, Elgin EE. Diffuse nonatherosclerotic coronary aneurysms. Cardiol Rev. 2005;13:309–311.
- c. Demopoulos VP, Olympios CD, Fakiolas CN, et al. The natural history of aneurismal coronary artery disease. Heart. 1997;78:136–141.
- d. Baman TS, Cole JH, Devireddy CM, et al. Risk factors and outcomes in patient with coronary artery aneurysms. Am J Cardiol. 2004;93:1549–1551.
- e. Sudhir K, Ports TA, Amidon TM, et al. Increased prevalence of coronary ectasia in heterozygous familial hypercholesterolemia. Circulation. 1995;91:1375–1380.
- f. Stone GW, Ellis SG, Cannon L, et al. Comparison of a polymer-based paclitaxel-eluting stent with a bare metal stent in patients with complex coronary artery disease: a randomized controlled trial. JAMA. 2005;294:1215–1223.
- g. Szalat A, Durst R, Cohen A, et al. Use of polytetrafluoroethylene-covered stent for treatment of coronary artery aneurysm. Catheter Cardiovasc Interv. 2005;66:203–208.