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Case Report Open Access

Spontaneous Regression of Subocclusive Coronary Stenosis: Does Time Heals All Wounds?

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Case Description

Surely the Beatles were not calling on interventional cardiologists in their last hit "Let it be", even though it would have been a good advice in the present clinical case. We report, for the first time, on the spontaneous regression of a subocclusive coronary stenosis over 11 years.

A 52-years old man, with hypertension, dyslipidemia and previous NSTEMI, was referred to our hospital for coronary angiography, which revealed a subocclusive stenosis of the proximal 3rd obtuse marginal branch (OM3), with antegrade TIMI 0/1 flow and retrograde perfusion through the LAD (1A,1B) (Figure 1). The patient refused any intervention and was therefore discharged on secondary cardiovascular prevention, including antiplatelet therapy with ASA 100 mg/die and clopidogrel 75 mg/die, a statin (simvastatin 20 mg/die) and a beta-blocker (atenolol 25 mg/die). The patient underwent a cardiologic control every sixth month. Clopidogrel was stopped some months later, while atenolol dosing was repeatedly modified depending on Blood Pressure (BP) and Heart Rate (HR) levels. Similarly, ramipril 5 mg/die and nitrates were prescribed for some time and stopped again during the follow up.

Eleven years later, the same patient was referred again to our hospital with the diagnosis of STEMI of the anterior wall after successful thrombolysis. Pharmacological treatment at presentation included ASA 100 mg/die, clopidogrel 75 mg/die, atorvastatin 80 mg/die, ramipril 5 mg bid, furosemide 25 mg/die, atenolol 50 mg/die. Coronary angiography revealed residual critical stenosis of the proximal LAD with thrombotic stratification which was successfully treated with stent-PCI. Very interestingly, the previously subocclusive OM3 stenosis was spontaneously regressed to a 70% stenosis with a TIMI III flow (1C,1D) (Figure 1). The patient was then discharged on the following therapy: ASA 100 mg/die, ticagrelor 90 mg b.i.d., atorvastatin 80 mg/die, ramipril 5 mg/die, furosemide 25 mg/die, atenolol 50 mg/die, spironolactone 25 mg/die. Importantly, the initial lesion was surely not a catheter-induced spasm, giving the distal position, nor any coronary wire had been inserted into the vessel. Furthermore, both angiograms were obtained after intracoronary nitroglycerin administration.

Thus the question remains: what was responsible for lesion regression in this patient? It is known that aggressive cholesterol lowering with statins slows progression of atherosclerosis, reduces new lesions' formation or even causes atheroma regression and prevents clinical events [1,2]. On the other hand the beneficial effects of increased HDL-C are also recognized [3].

Our patient had stable LDL-C levels. However HDL-C levels were substantially increased from the initial evaluation in 2002 throughout the follow up. In addition, a much better Blood Pressure (BP) control was achieved. Since the patient had significantly increased his physical activity after the initial evaluation in 2002, it is tempting to speculate this contributed to the observed improvement in HDL-C levels and BP control. Altogether, the increase in HDL-C levels, the improved BP control and the regular exercise could have contributed to the regression of the coronary stenosis.

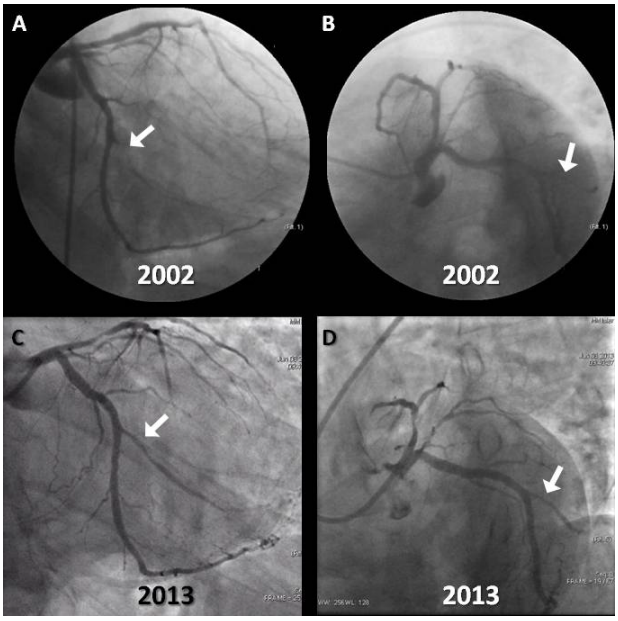

Figure 1: Regression of Atherosclerosis. **Panel A:** RAO view of the OM3 stenosis at the time of the first angiography, in 2002 showing subocclusive stenosis with incomplete filling of the downward vessel. **Panel B:** Spider view (caudal LAO) of the OM3 stenosis at the time of the first angiography, in 2002, confirming the finding of RAO. **Panel C:** RAO view of the same OM3 stenosis eleven years later (2013), showing stenosis regression with visualization of the whole downward vessel. **Panel D:** Spider view (caudal LAO) RAO view of the OM3 stenosis in 2013, confirming stenosis regression with contrast filling of the downward vessel.

In conclusion, the present report is the first one showing spontaneous regression of a subocclusive coronary artery stenosis. However, no conclusion can be reached on the underlying mechanisms.

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