

Ten Years After

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A famous novel which enchanted our youth was entitled *Vingt Ans Après* (*Twenty Years After*). It was written by Alexandre Dumas, to follow the destiny of his heroes in *Les Trois Mousquetaires* (*The Three Musketeers*).

Everything was optimistic after 20 years. D'Artagnan became officer in the army of his former enemy, Le Cardinal Richelieu, Athos came back alive to his home place, Porthos got married and Aramis became a priest.

Some 10 years before, I myself had a short look at what was identified to produce acute atherothrombosis (cerebral or myocardial infarction), a dramatic acute event associated with the

greatest figures of mortality in humans, and at how we could prevent it (1).

It was clear that the vulnerable atherosclerotic plaque was the enemy. For acute myocardial infarction (AMI), plaque rupture occurred and the acute thrombus blocked the coronary. In PubMed there were some 2 000 papers dealing with the subject.

Today, some 10 years after, I think we are not very far from what we knew, or could do, in 2015.

There were already many methods to identify an unstable plaque susceptible to rupture. Some of them were precise, but invasive and costly. Examples include intravascular ultrasound (IVUS), optical coherence tomography (OCT) or coronary angiography.

Other methods are non-invasive, but difficult to apply and expensive. Examples include computed tomography (CT), nuclear magnetic resonance (NMR) or positron emission tomography (PET) or even non-invasive high-definition thermography.

Today, none of the above-mentioned approaches are used to identify non-obstructive vulnerable plaques and treat them to prevent the acute event in clinics.

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But how to treat? Especially for prevention purpose?

At present, it is already decided that vulnerable plaques are prone to rupture when they have large lipid core in the plaque, a thin cap and inflammation (2).

Although reducing the LDL-cholesterol core is really possible, it has to be attacked long before the plaque develops towards a vulnerable one. Or in an artery tree there are tens of such candidate plaques in almost every person. Inflammation was attacked by colchicine, and even by antibodies like tocilizumab, ziltivekimab, canakinumab or anakinra receptor antagonists (3). However, none of them entered clinical practice to help treating.

The mechanical ways to cure an acute vulnerable plaque or event to prevent rupture are available in multiple variants.

No guidelines recommend preventive mechanical procedures to prevent rupture of an identified vulnerable plaque (4, 5).

So... 10 years after... only colchicine entered clinical practice... although so shyly, with a 2b B-R indication and – in my opinion – a negligible effect on major end points (4, 5).

It is said that progress in science... and cardiology is tremendously fast!

But to prevent the rupture of vulnerable plaques, we still have to wait. For some time... Five years? Ten years? Maybe longer? 



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