

Decision Arguments to Intervene in Coronary Artery Disease: Are We Serious ?

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A paradigm accompanies coronary artery pathology since decenies now: which is our main argument used when taking the decision how to make treatment ? This argument is the presence of ischemia.

The 70% stenosis or more is the most useful parameter. But why exactly 70%? Because starting from this point ischemia is generally presented. But not always. Than we may use the flow reserve. In practice is not used very often, because is costly.

It is indicated sometimes to search ischemia by different methods. The ECG stress test has

its limits, especially for sensitivity. Scintigraphic perfusion scan and echocardiography are costly (the first) or operator dependent and time consuming (the second).

It is logical to look for viable myocardium by different methods, but this is applicable only when heart failure accompanies coronary artery disease. Most of the coronary patients don't have heart failure.

But now comes the main question: is our main goal to treat chronic ischemia ? Most of the heart attacks come not because a severe stenosis is completing its progressive evolution, but because the plaque ruptures or becomes unstable and favors the development of an occlusive thrombus.

So, a patient with coronary arteries diseased may have or have not chronic ischemia. But for sure they are in danger of making acute ischemia when a thrombus adds to an existing plaque. And the main goal of therapy is not to treat chronic ischemia, but to treat or to prevent the acute thrombotic coronary phenomenon.

But the acute phenomenon has (almost) nothing to do with the previous presence of ischemia. Nobody demonstrated that at 70%, 80% and 90% stenosis are accompanied linearly or exponentially by a heart attack in the

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near future and the 60% stenoses are not (or significantly less often).

On the contrary: many pathological studies demonstrate that the ruptured or the unstable plaques originate mostly from less occlusive plaques, but with a soft composition which is unstable and may promote thrombogenesis.

Than why do not we use the diagnosis of an unstable plaque as the main argument to intervene promptly, instead to search for a significant stenosis? Because for the moment we do not know to identify with accuracy unstable plaques and therefore we use the surrogate end point of ischemia produced by a significant stenosis.

We may notice from this point of view that revascularization by interventional cardiology is an incomplete term. When implanting a stent, the plaque below is much more stabilized than before in most cases. If a restenosis occurs, this is done by a proliferative material much more stable than the original atherosclerotic plaque. We may say that by doing PTCA with stent we revascularize AND stabilize the atherosclerotic coronary.

So, our main indicator to intervene should be the instability of the coronary plaque. Can we identify it today?

Not yet, but in this direction progress is remarkable. In an excellent editorial in this issue of the Journal (1) Viviana Aursulesei makes a very good synthesis of the methods used currently in clinical research to identify the unstable plaque. Some methods seem now to be familiar to clinicians: intravascular ultrasound with virtual histology, high resolution magnetic resonance imaging, positron emission tomography and even optical coherence tomography have results in identifying unstable plaques

presented at general cardiology meetings and journals. Other methods, like palpografia, the elastogram or the modulogram of the plaque (1) are methods only known by a limited number of specialists.

The results of these methods are analyzed now in clinical trials with clinical outcome endpoints. The information given by the new methods are included in new risk factor charts to complete the traditional ones we use today. The accuracy of the new methods to predict acute events is profoundly analyzed.

The ability of the new indicators to predict coronary outcome is still the weak point. The sensibility and specificity of any of the new methods does not yet overcome the traditional way to predict bad outcome in coronary artery disease. Everything seems logical in the new parameters – however something is still missing to bring them in the pole position.

„Most likely, a multimarker strategy that includes risk factors, molecular and genetic biomarkers, noninvasive imaging markers, will be a truly viable option in terms of practice” – comments dr Aursulesei in her editorial and I totally agree.

Looking for ischemia instead of looking for the potential instability of a plaque seems to me to be a historical error of cardiology, assumed consciously until the moment when we will be capable to identify correctly the unstable plaque. Ischemia should stay in that moment as the second main endpoint of coronary disease therapy.

The presence of a potential unstable plaque is the real argument that we should be promptly aggressive in the therapy of coronary artery disease.

REFERENCES

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