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Editorials

Coronary angioplasty of rapidly progressive lesions — nothing to it or just too rare to be recognized as a problem?

See page 1671 for the article to which this Editorial refers

The paper from the Lille group in this issue[1] establishes a more than reasonable hypothesis only to refute it on the basis of their well documented data bank. They surmised that lesions having shown rapid progression before angioplasty have a higher preponderance for restenosis than those that built up more slowly. This hypothesis is based on data of their own group[2] and of others[3], revealing an increased restenosis rate in patients undergoing coronary angioplasty for unstable angina. They carefully avoid confusion with the high restenosis rate found after angioplasty for rapidly recurring lesions reported previously with data from the same data bank[4]. Such lesions are of different structure and pathophysiology (intimal proliferation and constrictive remodelling) whereas rapid progression of non-dilated lesions is based on progressive atherosclerosis and plaque rupture with organized thrombi[5]. Much to their and my surprise, the hypothesis fell short when scrutinized in their prospectively accumulated and carefully maintained data bank. Did the authors find the truth or did they fall victim to a type II error?

As often in biology, the answer is both 'yes' and 'no' to both questions. One can deduct conclusively from the paper that a rapidly progressive de novo lesion that is dilated during an elective procedure yields results comparable with those of lesions that have barely changed over the past 7 months. However, these quiescent lesions that have progressed recently but apparently have not been 'caught in the act' are not the ones we are truly worried about. Our concern in terms of acute problems and restenoses focuses on truly unstable lesions. Even in the group with rapid progression, only 20% of the patients were considered as unstable. This is lower than most series of unselected coronary angioplasty procedures. Two factors may account for the low percentage of unstable clinical presentations in a group of patients with documented progression of their coronary artery disease. First, the definition used for unstable angina (chest pain with electrocardiographic changes within 48 h before an elective coronary angioplasty) is quite stringent and somewhat contradictory. Second, the series is explicitly based on patients called in for a routine follow-up angiography at about 6 months after an initial successful coronary angioplasty. Through such a narrow window, unstable angina is rare to see. The authors then took a closer look at the patients with unstable angina (i.e. nine people) but were unable to confirm the high tendency of restenosis they had described earlier[2]. This was a type II error trap if ever there was one.

A number of additional points are of note in the report. The average reference vessel diameter of 2·5 mm of a reasonably large cohort (85 patients) of unselected coronary stenoses is small. This may be intrinsic to the method of quantitative coronary angiography employed. It may or may not influence the balloon selection. Again, the mean balloon diameter of 2·8 mm is clearly below industry standards. A certain systematic under-dilation cannot be excluded and may account for the relatively high degree of stenosis at the end of the procedure (32%) which, in turn, lends to a high categorical percentage of restenosis (not given in the paper) and a low late loss (0·3 mm). A low mean late loss will reduce the power of discrimination. A certain deficiency of the paper is the absence of data concerning the acute outcome of angioplasty in the two groups. The authors probably wished to prevent false conclusions based on type I or type II errors. In groups of only 40 patients, even in stentless procedures as the ones described, acute occlusion has only to be expected in about three patients per group. Of course, if the rapidly progressing group had only included patients with truly unstable angina, an increased incidence of acute problems could have become apparent. With a prevalence of unstable angina of 20% in the 'high

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risk' group and a mere 5% in the control group acute events are likely to have been very rare.

It is easy to concur with the authors that accidentally detected lesions that have recently progressed can and should be dilated without a significantly increased risk of recurrence. The community of angioplasty operators is grateful for these data, endorsing a long established practice. However, the paper does not minimize the acute problems or increased likelihood of restenosis when it comes to plaques dilated at the very moment of progression. The authors are far too experienced not to have conveyed this caveat clearly in the discussion.

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Arrhythmogenic right ventricular disease, dysplasia and cardiomyopathy

See page 1717 for the article to which this Editorial refers

This issue contains an interesting article on long-term changes in the ECGs of patients with ventricular arrhythmias originating in the right ventricle[1]. The authors describe their patients as having arrhythmogenic right ventricular disease but quote extensively references concerning arrhythmogenic right ventricular dysplasia. In fact the term arrhythmogenic right ventricular disease refers to a larger group of patients than the classical arrhythmogenic right ventricular dysplasia, which is clearly identified by its specific histological structure rarely available in a common clinical series[2].

In addition to the presence of ventricular tachycardia (of unknown origin) the authors' criteria for inclusion are based on contrast angiography. This is also an interesting approach in clinical diagnosis since angiography is strongly dependent on the underlying anatomical structure and has generally been considered as the 'gold standard' for the diagnosis by showing evidence of segmental abnormalities[3,4].

However, in addition to this approach the authors have included in their selection the so-called 'more diffuse forms'. We are concerned that these more diffuse forms may confuse the issue. Diffuse dilatation of the right ventricle alone is not a sign of arrhythmogenic right ventricular dysplasia. It could be the result of different forms of idiopathic cardiomyopathy mostly involving the right ventricle without replacement of myocardial fibres by fatty tissue[5]. Even segmental abnormalities could be the result of a localized form of healed myocarditis, ischaemia or cardiomyopathy[6]. Only specific segmental abnormalities, such as outpouching bulges, microaneurysms, or deep fissures in the infundibulum or at the apex are markers of arrhythmogenic right ventricular dysplasia. However, such comments are not of academic importance since the prognosis of these various subgroups may be different. For instance, the development of myocarditis recently demonstrated in a subgroup of cases superimposed on arrhythmogenic right ventricular dysplasia could lead to progressive modification of the ECG that is not the result of the dysplastic phenomenon (replacement of right ventricular musculature by adipocytes and fibrous tissue) but a different phenomenon that could affect both right and left ventricles[7]. Therefore, the changes observed in some of the cases may not be the result of evolution of the basic disease, but the consequence of a different phenomenon acting