investigations should include comparisons of the results of quantitative tomographic techniques such as positron emission tomography, with those of tomographic thallium scintigraphy.

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References


Angioscopic morphological changes after coronary angioplasty of unstable plaques

Angioscopy is one of several ways to assess the morphology of stenoses. In order to perform coronary angiography, a guiding catheter must be used to deliver the angioscope to the stenosis. Generally, guidewires are used to keep the angioscope coaxial with the blood vessel.

Angioscopy provides information about changes in the vessel lumen, but processes going on in the vascular wall underlying the lumen are not visualized by angioscopy. However, despite this limitation much can be learned from direct visualization of the stenoses, for example detailed characterization of the vessel lumen and morphological changes of the intima of the vessel.

Angioscopy can distinguish intracoronary thrombus from atheromatous tissue better than any other form of coronary artery visualization. It is the most sensitive method with which to detect coronary thrombus and can be used to classify atheromatous plaques[1]. In addition, coronary dissection is detected more accurately by angioscopy than by coronary angiography.

den-Heijer and colleagues support the position stated above with a study of 13 patients undergoing angioplasty[2]. They showed significant progression of intimal dissection and thrombus formation by angiography, which was not detected by angiography. They make the point that angioscopy has superior sensitivity to detect damage to the intima and thrombosis and by doing so can reveal important intravascular events that apparently occur even after successful angioplasty.

Coronary angiography will not replace angiography as a reference standard for imaging atherosclerotic coronary arteries[3]. However, angioscopy does offer a full colour, three-dimensional perspective of the intracoronary surface morphology. It is possible that visualizing details, not reliably available from angiography alone, may ultimately be used to assess risk and make clinical decisions at the time of angiography.

Limitations of angioscopy are: (1) the need to occlude the vessel during imaging which sometimes produces myocardial ischaemia; (2) there is presently no method to quantify angioscopy findings; (3) compared to intracoronary ultrasound the limitations are that angioscopy does not assess the cross-sectional image of the vessel and thus does not permit analysis of the layers of the vascular wall nor does it allow classification of the plaques related to plaque content e.g. thrombus, lipid, fibrous tissue and calcium.
One could ask "Is there a role for coronary angioscopy during routine coronary angiography?" If morphological characteristics of plaques compatible with future plaque rupture can be identified then perhaps specific therapy can be designed to prevent these plaques from rupturing and thus prevent myocardial infarction.

Uchida and colleagues provide some insight into the question of plaque morphology and prognosis\(^4\). They evaluated 157 patients with stable angina in whom regular coronary plaques were observed by percutaneous transluminal coronary angioscopy. Patients were followed for 12 months. Acute coronary syndromes occurred more frequently in patients with yellow plaques than in those with white plaque (11 of 39 vs 4 of 118; \(P=0.00021\)). These investigators pointed out that acute syndromes occurred more frequently in patients with glistening yellow plaques than in those with non-glistening yellow plaques (9 of 13 vs 2 of 26; \(P=0.0026\)).

In this issue, Bauters et al.\(^5\) report the first serial angioscopy studies on the effects of percutaneous transluminal coronary angioplasty on plaque morphology in 15 patients who underwent successful percutaneous transluminal coronary angioplasty for unstable coronary syndrome\(^6\). The procedure of angioplasty itself reduced the stenosis from an average of 74.5% to 31.6% diameter stenosis immediately after angioplasty. At follow-up the average percent stenosis was 52%. They were able to classify lesions as smooth concentric, smooth eccentric, complex, or ulcerated. In addition, they identified the colour of the plaque as either white or yellow. The morphological characteristics of the plaque immediately after percutaneous transluminal coronary angioplasty were variable but many showed complex dissection. In contrast, at follow-up all of the morphological characteristics were characterized as smooth concentric. The colour of the plaques following successful percutaneous transluminal coronary angioplasty were predominantly yellow whereas at follow-up (average 225 days) all but one were white. The authors do not mention this in the manuscript but I suspect that yellow plaques were associated with underlying lipid, whereas the white plaques were more consistent with hardened connective tissue, calcium, smooth muscle, etc. It is also interesting that no thrombus was seen in late follow-up yet this was fairly common immediately after percutaneous transluminal coronary angioplasty.

Results of their investigations demonstrate clear cut healing of unstable plaques in the months following angioplasty. An interesting finding in the patients undergoing late angiography was the fact that no significant difference was noted between those who had restenosis and those who had no restenosis.

It might be important to know what medical therapy was given to these patients following angioplasty. For example were all of these patients treated aggressively with lipid lowering agents thus possibly reducing the lipid content of any plaque, particularly one that was disrupted by angioplasty?

Perhaps the ultimate way to assess coronary vascular disease is with the combined use of coronary angioscopy, intravascular ultrasound and quantitative coronary angiography. If all three intravascular imaging modalities are used, it will be much easier to identify plaque tears, disruption and dissection, endothelial exfoliation, platelet aggregation, thrombosis at the angioplasty site and recoil of over stretched vessel wall segment.

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