Why are coronary plaques more malignant in the uraemic patient?

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It has recently been recognized that coronary plaques are not static obstructive lesions of the coronaries, but dynamic structures that can progress with time and are susceptible to rupture and coronary thrombosis. Cell activation and ensuing proteolysis cause destabilization of the fibrous cap at the interface between coronary lumen and the lipid deposits beneath the fibrous cap. Thus, the question arises whether the morphology of the coronary lesion in the uraemic patient with known high mortality from coronary disease [1], and possibly more aggressive atherogenesis [2] (although this point is controversial) differs from the non-renal patient. The figures show three findings which are characteristic of coronary arteries and the coronary plaque in the uraemic patient [3]. Firstly, the thickening of the media and intima of the diseased coronary artery is certainly more conspicuous than in the non-renal patient (Figure 1a vs Figure 1b), and this is confirmed by quantitative measurements. This finding is consistent with the previous observations of Ibels [4] in non-coronary vessels. Secondly, there is more pronounced calcification of the plaques (Figure 2) and this correlates with the mean serum phosphate concentration. X-ray diffraction analysis shows that the calcium containing deposits are hydroxyapatite. Thirdly, (Figure 3) the plaques are heavily infiltrated by activated macrophages, known forerunners of plaque destabilization and plaque rupture. For clinical nephrologists these findings are of interest since they may at least explain partly the malignant character of coronary plaques in the uraemic patient and the very frequent recurrence of coronary stenosis after percutaneous transluminal coronary angioplasty (PTCA) [5].

Suggested reading

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Fig. 1. (a) Representative coronary artery from a patient with uraemia (paraffin section, Elastica-van Gieson stain; magnification × 75). (b) Representative coronary artery from a non-renal patient (Paraffin section, Elastica-van Gieson stain; magnification × 75).

Fig. 2. Representative calcified plaque from an uraemic patient (paraffin section, Kossa stain; magnification × 100).

Fig. 3. Immunohistochemical staining of macrophages in a coronary plaque of an uraemic patient: note high density of macrophages in an ‘active’ plaque (paraffin section, immunohistochemistry using the CD 68 antibody; magnification × 100).