Branch retinal artery occlusion secondary to percutaneous transluminal coronary angioplasty

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SUMMARY We report on a patient suffering a branch retinal artery occlusion immediately following percutaneous transluminal coronary angioplasty. Balloon angioplasty may produce ocular embolic events consistent with previously reported neurological complaints.

Percutaneous transluminal coronary angioplasty (PTCA) was introduced in 1977.1 It has successfully opened obstructed coronary arteries in properly selected patients.2,3 Symptomatic, electrocardiographic, and angiographic improvement has been reported for up to three years after PTCA.4

The most frequent complications reported secondary to PTCA have been prolonged angina, myocardial infarction, and coronary occlusion.5 We report the first case of a branch retinal artery occlusive event secondary to PTCA.

Case report

A 62-year-old white male suffered an uncomplicated inferior wall myocardial infarction in June 1987 and developed post-infarction angina. Two months later, on 12 August 1987, he underwent successful PTCA for a 90% stenosed circumflex artery. Immediately following the PTCA the patient complained of a visual deficit in the left eye. Ophthalmic examination showed 20/20 vision in each eye. A central 5° inferior absolute scotoma was detected in the left eye on tangent screen visual field testing. Ophthalmoscopic examination of the fundus revealed a superior temporal branch artery occlusion with retinal oedema (Fig. 1).

Discussion

Distal emboli to the heart and kidney have been reported following both coronary and renal artery balloon angioplasty.6,7 In a study of 1500 patients following PTCA7 543 in-hospital complications (21%) were reported. No ocular complications were mentioned. Four neurological complications were reported, including one cerebrovascular event and three transient ischaemic attacks.8 These neurological complications and our patient’s branch retinal artery occlusion show that balloon angioplasty may produce embolic events affecting tissues other than the target organ.

There are two mechanisms of embolisation potentially responsible for the branch retinal artery occlusion in our patient. First, atherosclerotic debris originating from pre-existing vascular lesions can result directly from catheterisation. This process

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usually results in cholesterol emboli. Secondly, a well recognized type of embolisation in any form of percutaneous cardiac catheterisation is the result of thrombus formation on the exchange guidewires. Embolisation occurs during the guidewire exchange process. It is impossible to determine which process is responsible for the vascular occlusive event without irrefutable evidence that the embolisation was due to cholesterol.

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References


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