Haemorrhagic retinopathy in patients admitted with acute cardiac chest pain

Coronary heart disease is the most common cause of death in diabetic patients(1), and up to 25% of all patients presenting with acute myocardial infarction (AMI) are diabetic(2). Patients with AMI benefit from treatment with thrombolytic agents both in terms of reduced mortality and reduced morbidity(3). It has been stated that patients with diabetic retinopathy should not receive thrombolytic therapy because of the risk of intraocular bleeding(4); indeed there have been a few anecdotal reports of intraocular haemorrhage complicating thrombolytic therapy(5) but no prospective trials have systematically assessed this risk. Junior medical staff may have difficulty in obtaining adequate fundal views in patients who may have opiate-constricted pupils, and thus patients may not receive thrombolysis if there is doubt about the presence of diabetic retinopathy. This study reports a survey of retinopathy in patients passing through a coronary care unit, having presented with possible MI.

All patients presenting to the Coronary Care Unit at Seacroft Hospital over a 4-month period were eligible for inclusion in the study. Informed consent was obtained from all patients in the study, which was approved by the local ethical committee.

All patients had a history typical of cardiac chest pain (CCP) and ECG changes compatible with ischaemia or infarction. In all patients not known to be diabetic a formal glucose tolerance test (GTT) was carried out at day 3 and repeated after 6 weeks if abnormal. Visual acuities were checked, the pupils dilated and direct and indirect fundoscopy carried out, and where technically possible a retinal photographic survey was carried out using a 30° field retinal camera.

Sixty patients (41 male, 19 female) with a mean age of 65 years were admitted with CCP and 24 were given thrombolytic agents (23 streptokinase, 1 t-PA) during the study period.

Sixteen patients were diabetic and nine were hypertensive. Thirty patients had normal retinae. Nineteen patients had significant cataract, and three had retinal haemorrhage, two of whom had received thrombolysis. One of these patients was a newly diagnosed type II diabetic, with background retinopathy, whilst the other was not diabetic but had evidence of cholesterol embolus, with cotton wool spots and haemorrhage. There were no cases of vitreous haemorrhage or of altered visual acuity. The third patient had a flame-shaped haemorrhage in one eye, but was neither diabetic nor hypertensive.

This study demonstrates that in the routine practice of a CCU using thrombolytic therapy on a regular basis, the incidence of visual complications from thrombolysis is low, and there is no evidence that thrombolysis should be withheld from diabetic patients or patients in whom diabetes might be suspected on the grounds that there is significant risk to vision. This study does not address the risk to the small group of patients in whom there is proliferative retinopathy present at the time of chest pain for which thrombolysis might be given. The data suggest that a high proportion of patients presenting with CCP are in fact diabetic (16/60) and that the suggestion that diabetic patients should not be given thrombolysis should be doubly refuted on the grounds that the risk to vision is small if they are diabetic or not, and that to exclude diabetic patient from thrombolysis would be to deny treatment to a significant proportion of patients who might benefit from it most. This study shows that there can be retinal haemorrhage after thrombolysis which may not be associated with pre-existing retinopathy, or with non-diabetic retinal pathology. We believe the prospect that similar sub-clinical changes might occur in other tissues such as the brain following thrombolysis is intriguing but should not inhibit the use of a life-saving treatment.

M. SLEIGHTHOLM
P WANKLYN
M. KEARNEY
Seacroft Hospital, York Road,
Leeds LS14 6UH

References