ORIGINAL ARTICLES

Outcome of extensive coronary artery dissection during coronary angioplasty

Tim R Cripps, John M Morgan, Anthony F Rickards

Abstract

A total of 32 (3.6%) patients of 880 undergoing coronary angioplasty during a nine year period at one hospital had extensive dissection (defined as a dissection extending beyond the limits of the dilated angioplasty balloon) in the coronary artery in which the angioplasty procedure was performed. Two (6.25%) of the 32 patients (both of whom were undergoing angioplasty because of unstable angina that was refractory to medical treatment) died as a consequence of the coronary artery dissection. Twelve (38%) needed immediate coronary artery bypass surgery and 11 (34%) had a myocardial infarction, which in four was minor in extent. During follow up, 20 of the 32 patients were successfully managed by medical treatment; only two needed further angioplasty procedures. There were no late deaths. Extensive coronary artery dissection is a serious complication of coronary angioplasty, with a high early mortality and a high incidence of infarction and requirement for bypass surgery. None the less, patients with extensive dissection who are free from the manifestations of acute ischaemia at the end of the procedure can be managed conservatively and have a good immediate and medium term outlook. Attempts should be made to stabilise extensive dissection during coronary angioplasty so that surgical intervention can be delayed or avoided altogether if possible.

Coronary artery dissection is a well recognised complication of coronary angioplasty.¹ The dissection may be localised at the dilatation site or may extend distally along the length of the arterial lumen.

Though localised dissection and plaque splitting are probably important mechanisms underlying successful dilatation² most acute ischaemic complications of coronary angioplasty are associated with an intimal tear.³⁻⁵

Several studies have been directed towards establishing an association between the occurrence of localised dissection and vessel occlusion, but few have focused on major dissections that extend beyond the length of the angioplasty balloon.

Patients and methods

From 1980 to 1989, 880 patients were recorded as undergoing coronary angioplasty at the National Heart Hospital, London. A computer based search found that 32 patients had had extensive coronary artery dissection during the angioplasty procedure. This excluded those in whom dissection occurred during recanalisation of an occluded vessel ($n = 2$) or in whom dissection was caused during manipulation of the guiding catheter ($n = 1$).

Extensive dissection was defined as angiographic evidence of dissection extending beyond the segment of arterial wall that was contiguous with the fully dilated angioplasty balloon.

Of these 32 patients, 27 (84%) were men and 5 (16%) were women. Their mean age was 57 years (median 57 years, range 25-81). The procedure was performed in 25 (78%) patients with chronic stable angina, in five (16%) with unstable angina or evolving acute myocardial infarction, and in two (6%) for other reasons (after infarction in one patient aged 25 and during an evolving acute infarct in another).

Three patients had undergone one earlier coronary artery bypass operation; in one patient, surgery had been performed on two occasions. In three patients an earlier angioplasty had been performed.

Percutaneous transluminal angioplasty was performed with standard techniques and equipment. The femoral approach was used in all patients. β Blockers were stopped for 24 hours and aspirin given on the evening before the procedure. Intravenous heparin, 100 U/kg, was given routinely at the start and calcium antagonists were started after the procedure. Other antianginal agents were introduced subsequently as clinically indicated.

The policy on emergency surgery was as follows. When dissection occurred an attempt was made to "tack back" the dissection by long duration, low pressure inflations, often with an oversized balloon. When this approach was not successful, the patient was transferred for immediate surgery if there was continuing chest pain and electrocardiographic evidence of myocardial ischaemia, or haemodynamic instability. All patients who were managed medically had evidence of distal coronary flow at the end of the procedure.

Towards the end of the study period, balloon laser angioplasty and the intracoronary stent became available. These techniques were used only when the conventional tacking back

Royal Brompton
National Heart and
Lung Hospital, London
T R Cripps
J M Morgan
A F Rickards

Correspondence to
Dr Tim R Cripps,
Cardiac Department,
John Radcliffe Hospital,
Oxford OX3 9DU.

Accepted for publication
17 January 1991

Management of extensive dissection of the right coronary artery by laser balloon angioplasty. (A) Initial severe proximal right coronary artery stenosis (arrow indicates site of stenosis). (B) After a conventional dilatation procedure there was extensive dissection resulting in vessel occlusion (arrow indicates dissection flap; beyond this the distal vessel is replaced by a faint pool of extravascular contrast). The artery was reopened by low pressure dilatation with an oversized balloon but it closed again afterwards. (C) A laser balloon was then used to heat-weld the dissection flap at several points (arrows indicate dissection flap). Appearances were stable at restudy one week later. All the views are the left anterior oblique projection.

procedures had failed and where there was continuing ischaemia, in other words, in those patients who otherwise would have proceeded to emergency surgery.

Results

One artery was dissected in each of the 32 patients. The artery affected was the left anterior descending in 17 (53%), the right coronary artery in 11 (34%), the circumflex branch of the left coronary artery in three (9%), and a saphenous vein bypass graft to the right coronary artery in one (3%) patient.

Two patients (6.25%) died as an early consequence of the coronary arterial dissection despite early emergency bypass surgery. The two patients who died were clinically unstable before the procedure, with prolonged spontaneous ischaemic episodes unresponsive

to full medical treatment including intravenous nitrates; and one was elderly (73 years).

A diagnosis of myocardial infarction (based on the presence of at least two of the following: typical chest pain, new persistent electrocardiographic changes, or an increase in cardiac enzyme concentration above the upper limit of the reference range) was made in 11 (34%) patients. In four of them, however, the increase in serum enzyme was small and the infarctions were not associated with the development of new Q waves; the seven remaining patients had a Q wave infarction. Of those managed non-surgically after extensive dissection, only one had a Q wave infarct (inferior).

Immediate surgery was undertaken in 12 (38%) patients, because of continuing evidence of ischaemia, which could not be controlled by angioplasty techniques. The 12 patients included the two referred to above, who did not survive the operation. The remaining 10 patients survived the follow up period. Six of the 12 patients (including the two who died) who did have surgery had a Q wave infarction despite surgical intervention. This confirms that emergency surgery does not necessarily prevent myocardial infarction. In none of the patients was graft placement complicated by the presence of coronary artery dissection.

In all the patients, attempts were made first to stabilise the dissection medically, by the conventional approach of long duration, low pressure inflations, with an oversized angioplasty balloon. This was unsuccessful in all 12 of those who proceeded to emergency surgery

and in two others—one in whom a laser balloon angioplasty device (USCI) was used to heat-weld the dissection flap (figure) and another in whom an intracoronary self-expanding stent (Medinvent)⁶ was inserted to restore distal perfusion. In the remainder, tacking back was successful to a variable extent, but in all of them there was perfusion of the distal vessel at the end of the procedure.

Follow up continued (in those who survived the procedure and did not require surgery) for a mean of 19 months (median 7 months, range 1–50). No patient was lost to follow up, and no patient died during long term follow up. Two patients had elective surgery two months after the initial procedure for continuing disabling anginal symptoms that were unresponsive to medical treatment.

Of the 20 patients (63%) who survived the initial procedure and did not undergo surgery, three (15%) have grade III angina (Canadian Cardiovascular Society Classification) on medical treatment, but the other 17 have either no angina or minimal symptoms only.

Angiographic follow up was obtained in 12 patients. The figure shows an example of one such patient with a persistent stable coronary artery dissection. In seven of these 12 patients follow up angiography showed that the dissected vessel was patent.

Discussion

Extensive coronary artery dissection arising during the course of coronary angioplasty is a serious complication with a relatively high incidence of death, myocardial infarction, and the need for emergency coronary artery bypass surgery. We found, however, that in those patients with angiographically stable appearances with distal vessel perfusion and no ischaemic manifestations at the end of the procedure even extensive dissection was well tolerated in both the short and medium term so that immediate surgical intervention was not required.

It has been recognised for many years that localised dissection is a common finding after angioplasty, and that such localised intimal disruption usually heals well.⁷ Some degree of intimal disruption and plaque splitting may well underlie a successful angioplasty result.² None the less, in some patients dissection is associated with serious ischaemic complications including vessel occlusion.

Murphy *et al* found that 12 (70%) of 17 patients who needed surgery because of refractory ischaemia after angioplasty had coronary dissection.³ In another report 14 (70%) of 20 patients in whom acute occlusion developed after angioplasty had an intimal tear.⁸ In a report of 500 consecutive procedures performed in Geneva, there were 30 acute occlusions, of which 21 (70%) showed evidence of dissection at the end of the procedure.⁹ Bredlau *et al* reported a 6.5 fold increase in the risk of a major complication (emergency surgery, myocardial infarction, or death) when there was angiographic evidence of an intimal

dissection, while the major complication rate in those without dissection was only 1.6%.⁴

Shiu *et al* found angiographic evidence of dissection in six out of 20 patients with total occlusions, four of whom required surgery; one of those managed medically died, though the other had a successful result and repeat angiography showed healing of the dissection.¹⁰

Few previous reports focused on the consequences of major dissection, the angiographic appearances of which may be alarming (figure). In one such report, Black *et al* found that the dissection length was related to the occurrence of complications (ischaemic chest pain, myocardial infarction, need for bypass surgery, or death) late after the procedure (after the patient had left the laboratory), but their study included only seven patients of the type forming the basis of our report (tear > 15 mm) and four of them had ischaemic complications.¹

We found that two thirds (20/32) of the patients with extensive dissection survived without surgical intervention, with evidence of Q wave myocardial infarction in only one patient, who had an inferior infarct. None of these 20 patients managed without surgery required bypass surgery during follow up, and 17 now have no symptoms or mild angina only.

On the other hand, the seriousness of dissection should not be underestimated. The overall mortality in our series was 6.25%, and 38% of patients required bypass surgery. These figures are higher than would be expected for uncomplicated angioplasty procedures.⁴ Furthermore, though 20 patients survived to long term follow up, three have significant angina on full medical treatment and may require further intervention in the future.

Coronary artery bypass surgery can be complicated in patients with extensive coronary artery dissection by the need to re-establish the true lumen.¹¹ There is a risk of placing a graft in the subintimal plane. Bypass grafting was achieved without difficulty in all of our operated cases.

The conventional treatment for coronary artery dissection occurring during coronary angioplasty is to attempt to tack back the dissection flap by long duration low pressure inflations often with an oversized angioplasty balloon.^{12,13} This approach was successful in about half of our cases. In two patients in the later part of the series, however, novel approaches were used in patients who otherwise would have required surgery. In one, laser balloon angioplasty was used to stabilise the dissection and in the other an intracoronary stent was inserted. In both, the symptomatic and angiographic results were excellent and surgical intervention was avoided. The application of such novel methods may allow even more patients with extensive dissection to be managed conservatively. Immediate surgery for acute dissection is essential where ischaemia persists despite attempts to stabilise the patient non-surgically but in many cases it seems that it is possible to stabilise the dissection by angioplasty techniques, and in our experience the outcome is good in such cases. It may be that with increased use of adjuncts to conventional

angioplasty, including intracoronary stents⁶ and the laser balloon¹⁴ the proportion of patients requiring emergency surgical intervention can be further reduced.

Extensive coronary artery dissection is a serious complication of coronary angioplasty, with a high mortality, incidence of myocardial infarction, and requirement for immediate bypass surgery. However, most patients who suffer this complication seem to do well in the medium term with conservative management; this allows elective surgical intervention to be considered.

TRC was supported by the Charles Wolfson Charitable Trust.

- 1 Black AJR, Namay DL, Niederman AL, *et al.* Tear or dissection after coronary angioplasty. Morphologic correlates of an ischaemic complication. *Circulation* 1989;79:1035-42.
- 2 Block PC, Myler RK, Stertzer S, Fallon JT. Morphology after transluminal angioplasty in human beings. *N Engl J Med* 1981;305:382-5.
- 3 Murphy DA, Craver JM, Jones EL, *et al.* Surgical revascularization following unsuccessful percutaneous transluminal coronary angioplasty. *J Thorac Cardiovasc Surg* 1982;84:342-8.
- 4 Bredlau CE, Roubin GS, Leimgruber PP, *et al.* In-hospital morbidity and mortality in patients undergoing elective coronary angioplasty. *Circulation* 1985;72:1044-52.
- 5 Ellis SG, Roubin GS, King SB, *et al.* Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. *Circulation* 1988;77:372-9.
- 6 Sigwart U, Puel J, Mirkovitch V, Joffe F, Kappenberger L. Intravascular stents to prevent occlusion and restenosis after coronary angioplasty. *N Engl J Med* 1987;316:701-6.
- 7 Dorros G, Spring DA. Healing of coronary artery intimal dissection after percutaneous transluminal angioplasty [Abstract]. *Am J Cardiol* 1980;45:423.
- 8 Hollman J, Gruentzig AR, Douglas JS, *et al.* Acute occlusion after percutaneous transluminal coronary angioplasty—a new approach. *Circulation* 1983;68:725-32.
- 9 Steffenino G, Meier B, Finci L, *et al.* Acute complications of elective coronary angioplasty: a review of 500 consecutive procedures. *Br Heart J* 1988;59:151-8.
- 10 Shiu MF, Silverton NP, Oakley D, Cumberland D. Acute coronary occlusion during percutaneous transluminal coronary angioplasty. *Br Heart J* 1985;54:129-33.
- 11 Phillips SJ, Kongtahworn C, Zeff RH, *et al.* Disrupted coronary artery caused by angioplasty: supportive and surgical considerations. *Ann Thorac Surg* 1989;47:880-3.
- 12 Marquis J-F, Schwartz L, Aldridge H, *et al.* Acute coronary artery occlusion during percutaneous transluminal coronary angioplasty treated by redilation of the occluded segment. *J Am Coll Cardiol* 1984;4:1268-71.
- 13 Simpendorfer C, Belardi J, Bellamy G, *et al.* Frequency, management and follow-up of patients with acute coronary occlusions after percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1987;59:267-9.
- 14 Hiehle JF, Bourgelais DBC, Shapshay S, *et al.* Nd-YAG laser fusion of human atheromatous plaque-arterial wall separations in vitro. *Am J Cardiol* 1985;56:953-7.