Acute coronary occlusion during coronary angiography in two cases

Treatment by transluminal disobliteration

A CRIBIER, J BERLAND, G BRUNHES, C RICHARD, B LETAC

From Service de Soins Intensifs Cardiologiques et des Explorations Hémodynamiques Cardiovasculaires, Centre Hospitalier et Universitaire, Hôpital Charles-Nicolle, Rouen, France

SUMMARY Acute coronary obstruction occurred in two patients during coronary angiography. In one case the obstruction was in the left main coronary artery; in the other it was close to the origin of the left anterior descending artery. In both cases acute cardiac ischaemia ensued, with electromechanical dissociation and collapse, which was not reversible by resuscitation. Rapid disobliteration of the occluded coronary artery was done with a guide-wire pushed through the obstruction via the coronary catheter. The recanalisation was completed by an intracoronary perfusion of streptokinase in one case. In both cases recovery was rapid and spectacular. The occurrence of acute ischaemia during coronary angiography should suggest accidental coronary occlusion. If a thromboembolic origin is suspected, transluminal disobliteration should be attempted. It is simple and can reverse a dangerous condition.

The prevention of acute thromboembolic accidents during arterial catheterisation must be a constant concern of the operator. In the case of selective coronary angiography this risk is increased by the possibility of coronary embolism, the consequences of which can be dramatic: this accident is one of the main causes of death during this investigation.

Despite the experience of our team of 10 years of intensive cardiovascular exploration (with more than 1 000 investigations in 1980 alone), most of the cases including coronary angiography, the "law of series" was such that two coronary obstructions occurred at a few days' interval in our laboratory. In both cases there was acute irreversible shock and this led us to attempt coronary disobliteration according to the technique recently proposed in cases of acute myocardial infarction.¹⁻³

Case reports

CASE 1

A 60-year-old woman had a posteroinferior myocardial infarction in September 1979. The persistence of severe angina pectoris led to coronary angiography. The investigation was done on 27 May 1980 by the femoral transcutaneous method. Selective left ven-

tricular angiography, through a pigtail 7 F Cook catheter, showed a ventricular cavity with a normal volume, posteroinferior akinesia, and a contractile anterior wall, ensuring satisfactory ventricular function. The left coronary tree was seen during this injection, but not the right. The catheter was then changed for a Bourassa 8 F left coronary catheter. The coronary ostium was easily reached and was not occluded by the catheter as shown by control of the continuously monitored aortic pressure curve. In the seconds following the first injection, the patient complained of severe thoracic pain while the contrast medium only opacified the first centimetre of the left main coronary, seemingly abutting against an obstacle (Fig. 1a). The electrocardiogram immediately showed major anterior ST segment elevation, followed by extreme bradycardia, cardiovascular collapse, and loss of consciousness. Cardiac massage and other resuscitative measures were begun.

Twenty minutes later the patient's condition was still critical and it was decided to attempt coronary disobliteration with a guide-wire. The catheter was then changed and the left main coronary artery was again reached with another 8 F Bourassa catheter. Glyceryl trinitrate, 2 mg, was then injected into the left coronary artery, but there was no recanalisation on a subsequent angiogram. A straight Teflon guide-wire,

uncored for the last 8 cm (Cordis J guide, 0.35 mm diameter), was then pushed through the catheter into the left main coronary artery, first into the left anterior descending artery approximately to its mid-course (Fig. 1b), and then into the first 3 or 4 cm of the circumflex artery. The guide-wire was removed a few seconds after its intracoronary insertion and a subsequent injection of contrast medium showed the whole of the left coronary arterial bed (Fig. 1c). There were two areas of severe stenosis on the left anterior descending and circumflex arteries which explain the limited progression of the guide-wire. During this injection, ventricular fibrillation occurred, whereas the electrocardiogram had shown only occasional broad complexes since the onset of the resuscitative measures. After three cardioversions, sinus rhythm reappeared and blood pressure immediately returned to initial values. A few minutes later, spontaneous breathing resumed and the patient regained consciousness. Twenty minutes after recanalisation, while lignocaine (2 mg/min) was perfused, another injection of contrast medium confirmed the patency of the left coronary arterial tree.

Her electrocardiogram having returned to normal, the patient was transferred to the intensive care unit where heparin treatment was continued, lignocaine being stopped after a few hours. Enzymatic assays showed an increase of CK to 900 IU with an MB fraction of 3.5%, presumably because of the cardiac massage and cardioversions, and possibly cardiac reperfusion and myocardial damage. After 10 days in

hospital, without any chest pain or electrical change, the patient was discharged on beta-blockers. Another coronary angiogram was arranged two months later but this was refused by the patient.

CASE 2

A 67-year-old man had mitral regurgitation of long standing and was investigated on 4 June 1980 because of a recent episode of acute pulmonary oedema. Right heart pressure measurements confirmed unsatisfactory haemodynamics with a pulmonary systolic pressure of 60 mmHg, and a mean pulmonary wedge pressure of 28 mmHg. The arterial catheterisation started with a femoral transcutaneous puncture, followed by selective left ventricular angiography (Cook 7 F Pig-tail catheter). The left ventricle was moderately dilated and globally hypocontractile, and mitral regurgitation was severe. Right and left coronary arteries were weakly opacified but the first centimetres of the left anterior descending artery were clearly seen. After ventriculography the Cook 7 F catheter was replaced by a left coronary 8 F Bourassa catheter. This catheter was the wrong shape and it was replaced by another of the same type. The tip of this catheter was positioned by the left coronary ostium, but before any contrast medium could be injected the patient complained of severe constricting chest pain and the electrocardiogram showed significant ST segment elevation in the anteroseptal leads. Extreme bradycardia and cardiovascular collapse followed and the blood pressure remained extremely low

Fig. 1 Angiograms with explanatory diagrams. A, Coronary catheter in the ascending aorta, with its tip in the left coronary ostium; B, obstructed left main coronary; C, guide-wire pushed into the left anterior descending artery; D, revascularised circumflex artery; E, severe stenosis on the left anterior descending artery.

despite right ventricular electrical pacing. The alteration of the patient's consciousness and the inefficacy of a dopamine perfusion suggested that his condition was irreversible.

It was decided to obtain an angiogram of the left coronary artery despite the fact that the heart appeared immobile on fluoroscopy. The catheter was changed and yet another Bourassa left coronary catheter was introduced. Opacification showed an obstruction of the anterior descending artery close to its origin from a normal left main coronary (Fig. 2a). The circumflex artery was normal except for severe stenosis at the origin of its obtuse marginal branch.

The intracoronary injection of glyceryl trinitrate, 3 mg, had no effect. A Cordis J guide (diameter 0.35 mm) was then introduced into the left main coronary artery and easily pushed for 6 or 7 cm into the left anterior descending artery (Fig. 2b). Ventricular fibrillation occurred when the guide-wire was removed; the electrocardiogram had previously shown only considerably widened electrostimulated complexes.

Cardioversion promptly restored sinus rhythm, at which time left ventricular contractility reappeared and the systolic blood pressure rose to 120 mmHg.

Another angiogram of the left coronary confirmed the recanalisation of the left anterior descending

artery and of its branches. The opacification of this recanalised vascular tree, however, appeared distinctly later than that of the circumflex, suggesting that the disobliteration had been incomplete. The catheter tip was advanced to the origin of the left anterior descending artery and streptokinase, 20 000 units, was slowly injected through the catheter for two minutes. Just after this, another angiogram showed symmetrical flow in both left coronary branches, confirming improvement in the quality of the recanalisation (Fig. 2c). After these procedures the patient's clinical state was satisfactory, without chest pain, and with a stable blood pressure. Electrocardiograms showed persistence of ST segment elevation in leads I and aVL. He was transferred to the intensive care unit where lignocaine, 2 mg/min, was given for 24 hours, in addition to heparin and digitalis and diuretic treatment. CK rose to 660 IU, with a 7% MB fraction, the increase in enzyme levels presumably being the result of a high lateral necrosis which was confirmed by the subsequent appearance of a Q wave in lead I and aVL. After good clinical progress the patient was discharged two weeks later. At a second investigation before mitral valve replacement, two months after this, the patency of the left coronary tree was confirmed. There was a severe stenosis at the origin of the anterior branch of the circumflex, and another less

Fig. 2 A, Coronary angiography catheter in the ascending aorta, its tip in the left coronary ostium; B, obstructed left anterior descending artery; C, circumflex artery; C', obtuse marginal branch; D, guide-wire pushed into the left anterior descending artery; E, severe stenosis at the origin of obtuse marginal branch; F, revascularised diagonal branch; G, revascularised left anterior descending artery.

The right ventricular pacing catheter and the part of the catheter in the ascending aorta which are visible on the photographs are not shown on the diagrams.

severe stenosis at the origin of the left anterior descending artery.

Successful valvular replacement and aorto-circumflex bypass grafting followed.

Discussion

Coronary angiography, though routinely performed, is not totally devoid of danger. Published reports indicate a number of complications and death is not exceptional, occurring in approximately 0.4% of cases, the figure varying considerably.⁴⁻¹¹ These variations seem to depend at least in part on the experience of the operators and the techniques used but also on whether or not deaths occurring up to 48 hours⁸ or even 10 days later¹¹ are included.

Acute myocardial ischaemia leading to death from cardiogenic shock and electromechanical dissociation is a serious complication in coronary angiography, death often occurring during the examination itself.⁵ Probably in most cases this is caused by acute coronary occlusion, the angiographic criterion of which is the sudden obstruction of a previously opacified patent coronary artery. In some cases this can result from a traumatic intimal dissection,^{5 12} from persistent spasm,⁷ or, classically, from the mobilisation of an atheromatous plaque, but in most cases it is the result of thrombus originating from the catheter. In an angiographic and anatomical study of 66 deaths occurring during coronary angiography, Takaro *et al.*¹¹ found this to be the case in 35 patients, that is 53% of their cases. In most series however, this cause, though strongly suspected, is not proven.^{4 10 13} Bourassa and Noble⁵ found a fresh intracoronary thrombus only twice and histological evidence for recent infarction only three times in nine necropsies on patients who had developed sudden ischaemia during coronary angiography.

In our two cases, the diagnosis of intracoronary thrombosis was strongly suspected on angiography by the appearance of abrupt termination of the left coronary artery. In both, the total absence of effect of intracoronary glyceryl trinitrate probably excluded spasm. The possibility of coronary dissection may be entertained in the first case despite the lack of any typical angiographic evidence, but is not plausible in the second, since the obstruction was several centimetres away from the injection site.

The increased thromboembolic risk of arterial catheterisation by the femoral compared with the brachial route is well documented^{4 9-11 14} and could be the result of the use of guide-wires. When the guide is withdrawn from the catheter, the thin layer of platelet aggregates rapidly formed upon it¹⁵ can adhere externally to the catheter tip and then become detached and embolise. This may pass into a coronary

ostium, thus causing a coronary obstruction, more often at the site of a proximal severe stenosis.^{5 11} Prophylactic intravenous heparin is recommended^{10 13 16} but does not eliminate all thromboembolic events, as shown by Bourassa and Noble⁵ and as our two cases confirm, since our patients all receive an intravenous dose of 50 mg heparin at the beginning of the procedure.

Though it has sometimes been attempted,^{5 10 11 17} emergency aortocoronary bypass is almost always doomed to failure in such instances. In our two cases the simple act of pushing a guide-wire through the obstruction, with or without the intracoronary injection or infusion of fibrinolytic drugs, appeared to be the only possible course of action. We had already had experience with this technique in more than 30 cases of acute myocardial infarction, using the method proposed by Rentrop *et al.*¹ The efficacy of this action in both cases was plain. In the first patient, the guide-wire produced perfect angiographic recanalisation of the left coronary artery and a subsequent injection of streptokinase was not necessary. The immediate result excluded the hypothesis of spontaneous lysis of the clot. In the second patient, the guide passed through the obstruction but a noticeable delay in the opacification of the distal coronary bed persisted and an injection of streptokinase improved distal perfusion.

The clinical improvement of both patients after disobliteration was rapid and spectacular. In both, the removal of the guide-wire was followed by ventricular fibrillation which can be interpreted as the consequence of myocardial revascularisation. No electrocardiographic sequelae of the accident occurred in the first case; in the second the subsequent high lateral infarction was possibly the result of the obstruction of the stenosed circumflex branch not reached by either the guide-wire or the streptokinase.

Thus, the occurrence of sudden myocardial ischaemia during coronary angiography may be caused by accidental coronary occlusion. However critical the clinical state may be, it is essential to define the site and type of the obstructive lesion by a selective injection of contrast medium while resuscitative measures are continued. If thromboembolism is suspected, it is legitimate to try transluminal recanalisation. The technique is simple and can produce a dramatic reversal of an apparently hopeless situation.

The authors wish to thank Mr N Moore for his assistance in the translation of this manuscript.

References

- 1 Rentrop P, Blanke H, Kosterling H, Karsch KR. Intrakoronare Streptokinase-Applikation beim akutem infarkt

- und instabiler angina pectoris. *Dtsch Med Wochenschr* 1980; 105: 221-6.
- 2 Mathey DG, Kuck KH, Tilsner V, Kribber HJ, Bleifeld W. Nonsurgical coronary artery recanalization in acute transmural myocardial infarction. *Circulation* 1981; 63: 489-97.
 - 3 Ganz W, Buchbinder N, Marcus H, et al. Intracoronary thrombolysis in evolving myocardial infarction. *Am Heart J* 1981; 101: 4-13.
 - 4 Adams DF, Fraser DB, Abrams HL. The complications of coronary arteriography. *Circulation* 1973; 48: 609-18.
 - 5 Bourassa MG, Noble J. Complication rate of coronary arteriography. A review of 5250 cases studied by a percutaneous femoral technique. *Circulation* 1976; 53: 106-14.
 - 6 Anonymous. Hazards of coronary arteriography (Editorial). *Br Med J* 1971; ii: 3.
 - 7 Engel HJ, Page HL Jr, Campbell WB. Coronary artery spasm as the cause of myocardial infarction during coronary arteriography. *Am Heart J* 1976; 91: 501-6.
 - 8 Delaye J. Le risque de la coronarographie dans l'angor instable. *Arch Mal Coeur* 1979; 73: 211-5.
 - 9 Schoonmaker FW, King SB. Coronary arteriography by the single catheter percutaneous femoral technique. *Circulation* 1974; 50: 735-40.
 - 10 Shah A, Gnoj J, Fisher VJ. Complications of selective coronary arteriography by the Judkins technique and their prevention. *Am Heart J* 1975; 90: 353-9.
 - 11 Takaro T, Hultgren HN, Littmann D, Wright EC. An analysis of deaths occurring in association with coronary arteriography. *Am Heart J* 1973; 86: 587-97.
 - 12 Green GS, McKinnon CM, Rosch J, Judkins MP. Complications of selective percutaneous transfemoral coronary arteriography and their prevention. *Circulation* 1972; 45: 552-7.
 - 13 Judkins MP, Gander MP. Prevention of complications of coronary arteriography. *Circulation* 1974; 49: 599-602.
 - 14 Petch MC, Sutton R, Jefferson KE. Safety of coronary arteriography. *Br Heart J* 1973; 35: 377-80.
 - 15 McCarty RJ, Glasser SP. Thrombogenicity of guide-wires. *Am J Cardiol* 1973; 32: 943-6.
 - 16 Eyer KM. Complications of transfemoral coronary arteriography and their prevention using Heparin. *Am Heart J* 1973; 86: 428.
 - 17 Scanlon PJ, Nemickas R, Tobin JR Jr, Anderson W, Montoya A, Pifarre R. Myocardial revascularization during acute phase of myocardial infarction. *JAMA* 1971; 218: 207-12.

Requests for reprints to Professor B Letac, Service des Soins Intensifs Cardiologiques et des Explorations Hémodynamiques Cardiovasculaires, Hôpital Charles-Nicolle, 1 rue de Germont, 76000 Rouen, France.