Spontaneous coronary artery perforation with tamponade

Demonstration by necropsy selective coronary arteriography

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SUMMARY Pericardial tamponade in a 51-year-old man after acute antero-septal myocardial infarction was the result of spontaneous perforation of an atheromatous coronary artery. The value of a simple necropsy technique of selective coronary arteriography in demonstrating the lesion is described.

Cardiac tamponade from haemopericardium after acute myocardial infarction is usually caused by left ventricular perforation through the necrotic centre of a transmural infarct and less often by an anticoagulation pericardial bleed. In this report necropsy selective coronary arteriography was used to show that it can also be caused by perforation of an atherosclerotic coronary artery.

Method

Necropsy coronary arteriography was carried out as follows. The coronary arteries of the excised unopened heart were injected individually under image intensification using occasional large film exposures in addition to frequent serial single 70 mm camera shots. Exposures were first made of the uninjected heart in the right anterior oblique and left lateral views to identify calcification of coronary arteries later to be obscured by contrast. The aorta was cut transversely about 15 mm above the coronary orifices which were carefully examined, and the main coronary arteries were palpated in their external length for calcification and gross atheroma. Surgical coronary infusion catheters were tied into the orifices with purse string sutures and each coronary artery was injected in turn under an image intensifier. Using hand injection, Urografin 76 was instilled, very gently at first, with a 10 ml syringe. Frequent spot 70 mm films were taken as the contrast slowly filled the main coronary artery and then its branches. The force of hand injection was carefully increased so that branches and collaterals were eventually filled, serial spot films being taken to identify partial or complete occlusion. Exposures were repeated in another view before contrast was flushed from the coronary tree and

chambers with warm water. The same injection and exposure technique was performed on the other artery, care being taken to fill both left main branches.

Case history

A sheet metal worker aged 51 with no previous symptomatic vascular disease developed a localised pain to the left of his sternum worsened by inspiration and fairly constant for 36 hours when it became much more severe and he was admitted to the coronary care unit. He smoked 30 cigarettes daily.

On admission, the heart rate was 100 per minute and regular with a fourth heart sound. Blood pressure was 180/80 mmHg. Electrocardiogram showed right bundle-branch block, left anterior hemiblock, and an acute antero-septal injury, followed by infarct pattern. Enzyme peak levels were SGOT (AST) 520 units (normal 12-40 units/ml) and CK 1140 units (normal 0-100). X-ray film showed slight prominence of the left ventricle, with no significant cardiac enlargement but considerable dilatation and unfolding of the ascending aorta. Lung vascularity was normal.

By the second day there was bilateral basal consolidation and pericardial friction as well as a third heart sound. He was given anticoagulants from the time of admission and 40 mg frusemide was administered daily with Slow-K. Monitoring was stopped on the fourth day but pericardial friction was still present on the sixth day when he was transferred to an aftercare ward. On the following day heart rate was 80 per minute. Warfarin anticoagulant control was satisfactory (BCR ratio 1.8. Therapeutic range 1.5 to 2.5) and his condition seemed stable despite the persisting pericardial and

lung base signs. Late that night he had an apparent cardiac arrest while sitting on a commode and was resuscitated by praecordial thump delivered by the night nurse. Nodal rhythm around 100 per minute was identified but he quickly lapsed into unconsciousness. Subsequently the portable oscilloscope tracing showed a regular but gradually slowing agonal ventricular rhythm until he was clinically dead. Jugular venous distension was not reported at any stage.

RESULTS AT NECROPSY

The pericardial sac was tense, containing 425 ml blood and clot. Also present was some fibrinous visceral pericarditis and a degree of atheromatous change, with some ulceration, affecting the aortic arch and straight branches. No ventricular perforation was found. The lungs showed only basal congestion.

At necropsy coronary arteriography, plain views of the heart in the right anterior oblique view showed flecks of calcification in the region of the left anterior descending branch. Gentle palpation with a soft 'teflon' probe disclosed gross craggy disease within 15 mm of the main left orifice. Selective injection of contrast showed that the right coronary artery was grossly diseased, with irregular narrowing throughout its main length and good filling of branches but no evidence of collateral filling even with high finger injection pressure (Fig. 1). The left coronary arteriogram showed satisfactory filling of circumflex branches. There was gross atheroma of the initial 25 mm of the left anterior descending

branch, with almost complete obstruction. On higher pressure injection there was minimal filling beyond this and a jet of dye emerged from a point in the narrowed area, spreading out as a mushroom cloud at the surface of the ventricle (Fig. 2).

On examination of the heart after coronary angiography had been performed, no coronary arterial perforation was obvious. The presence of a recent anteroseptal infarct affecting the full thickness of the ventricular wall was confirmed. The left anterior descending branch showed diffuse atheroma with subsequent narrowing of the lumen, and beyond this antemortem thrombus, the histological appearance of which correlated well with the clinical history. Detailed examination of the artery between 26 mm and 30 mm from its origin showed evidence of dissection (Fig. 3). At one point in the circumferential atheromatous plaque (ap), the endothelium was breached (b) and a narrow fibrin lined track (t) led right through the media (m) to the adventitia (a) without showing a significant haemorrhagic collection.

Discussion

The patient died with tamponade from haemopericardium without ventricular perforation or pericardial surface bleeding. The cloud of dye which emerged from the wall of the left anterior descending branch during higher syringe pressure suggests that blood had been jetting into the pericardium during systole. In such a case, tamponade might be expected to develop slowly and if recognised

Fig. 1 Necropsy right coronary arteriogram (right anterior oblique position) showing gross narrowing of the main right coronary artery (RCA). SNA, sinus node artery; RV, right ventricular branches; Ca, calcified segment of left anterior descending branch (LAD); P, Pin marking the line of the LAD.

clinically to be particularly amenable to surgical management.

At necropsy, this method of simplified coronary arteriography has the advantage of speed, so that the heart may be returned to the pathologist in an undamaged state for further macroscopical and histological study. Films taken before injection will show any calcification of valves and of coronary arteries. The technique will portray the extent of coronary artery disease at least as well as selective coronary arteriography in life, though not with the pristine clarity seen in Fulton's painstaking work (1965), using bismuth gelatine and stereoradiography. It can show patency of collaterals and may indicate why collaterals have not opened up or are shut down. It can be used to show patency of and 'run off' from a saphenous vein graft and, by injecting only the distal segment of a proximally occluded artery, it can help to determine whether a graft attempt might have been successful. Aneurysms of coronary arteries both fusiform and saccular are clearly outlined by the method. It may disclose dissection of a coronary artery and, as in the present case, it is probably the only method of showing spontaneous perforation of a coronary artery.

Reference

Fulton, W. F. M. (1965). *The Coronary Arteries*. Charles C. Thomas, Springfield, Illinois.

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Fig. 2 Necropsy left coronary arteriogram (left anterior oblique position) showing the cloud of contrast (CC) jetting from proximal segment of left anterior descending branch (LAD) where there is gross atheromatous narrowing. P, marker pin; CIRC, left circumflex branch.

Fig. 3 A transverse histological section of the left anterior descending coronary artery with the fibrin lined track (t) leading from the breach (b) in the endothelium through the atheromatous plaque (ap) and the media (m) to the adventitia (a).