Left mainstem coronary artery ostial stenosis — death after angiography

A report of 5 cases

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Summary

Death directly related to selective coronary arteriography in 5 patients with a history of unstable angina pectoris during the period 1975 - 1985 is reported. Four different cardiologists were involved.

A feature common to all the cases was the presence of significant ostial stenosis of the left mainstem coronary artery (LMCA); 2 patients had haemodynamically important obstruction of a dominant right coronary artery (RCA) ostium, while 2 others had total occlusion in the proximal part of a dominant RCA. The RCA in the last case was angiographically normal and non-dominant. Collateral coronary blood flow was fairly sparse in most cases and in 4 left ventricular dysfunction of varying degree was present. All patients developed severe hypotension and electromechanical dissociation after arteriography while still in the cardiac catheterization laboratory. Resuscitation efforts were uniformly unsuccessful. Autopsy on 1 patient demonstrated extensive obstructive coronary atherosclerosis with a massive acute anterior myocardial infarction.

Cardiac catheterization poses an extremely high risk for this subgroup of patients with LMCA disease, as does selective coronary arteriography. The possible role of catheter-provoked coronary vasospasm of the LMCA is suggested; a recently introduced soft-tipped cardiovascular catheter may be more appropriate in this setting.

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Case reports

Case 1

A 47-year-old white man had a history of classic effort-related angina pectoris for 8 months. Approximately 2 months before admission to the Intensive Coronary Care Unit (ICCU) at Tygerberg Hospital, he had noted increasing frequency and severity of pain, as well as angina at rest. His general practitioner started therapy with β-blockers, calcium antagonists and nitrates, but this did not provide much relief, and he was referred for coronary arteriography with a view to possible surgery. His only risk factor for ischaemic heart disease (IHD) was that he smoked 10 - 20 cigarettes daily.

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Clinical examination revealed a healthy-looking man with no obvious features of hyperlipidaemia. His blood pressure was 110/70 mmHg and his heart rate 72/min and regular; there were no signs of cardiac failure. The rest of the physical examination yielded normal results. A resting ECG documented sinus rhythm of 61/min, a P-R interval of 0,15 second, a mean QRS axis of +40°, and horizontal ST segments without depression in the lateral leads. A chest radiograph was normal. Biochemical and haematological values were all within normal limits.

Cardiac catheterization was performed on 19 October 1979. The left-sided intracardiac pressures were normal. Left ventricular (LV) cine angiography in the right anterior oblique (RAO) projection demonstrated a normally contracting chamber with no evidence of mitral insufficiency. Selective coronary angiography delineated a dominant and diffusely diseased right coronary artery (RCA), totally occluded in its first part and with some right-to-right collateralization. The left coronary artery (LCA) had a subtotal ostial stenosis of the left mainstem with extensive left-to-left collateral blood flow (Fig. 1). There was no coronary calcification. Soon after completion of the procedure, and while the patient was still on the cardiac catheterization table, he complained of severe precordial pain. He was immediately given sublingual nitrate and nifedipine and oxygen but the pain did not abate and was followed by ventricular tachycardia which degenerated into ventricular fibrillation. Electrical defibrillation was performed several times and anti-arrhythmic drugs were given. Sinus bradycardia with hypotension ensued, necessitating insertion of a temporary transvenous cardiac pacemaker and inotropic therapy. However, the hypotension could not be reversed and electromechanical dissociation resulted. The resuscitation was discontinued after approximately 1 hour. Autopsy was not performed. The cause of death was considered to be a massive acute anterior myocardial infarction.

Case 2

This 71-year-old white woman began to experience stable angina pectoris in 1959. This pattern changed to one of unstable angina pectoris in 1976, at which time a resting ECG showed 'left ventricular hypertrophy with strain pattern'. At this time she was taking moderate doses of propranolol and sublingual nitrates when required. Four years later, in early 1980, she was admitted to the ICCU at Tygerberg Hospital with a diagnosis of unstable angina pectoris.

Physical examination revealed signs of hyperlipoproteinaemia in a fairly obese woman. Her blood pressure was 130/90 mmHg and pulse rate 56/min. There was no cardiomegaly. A 4th heart sound was audible and an aortic nodular sclerosis murmur was noted. There were no features of cardiac failure. A resting ECG highlighted asymmetrical T-wave inversion and 1 mm downward-sloping ST-segment depression in the apical and inferolateral leads. Chest radiography demonstrated borderline LV cardiomegaly and lung fields suggestive of early heart failure. Routine blood investigation results and serial serum enzyme levels were all normal.

Cardiac catheterization on 11 April revealed elevated left ventricular end-diastolic pressure (LVEDP) of 24 mmHg and normal central aortic pressure. Mild pulmonary hypertension (35/16 mmHg with a mean of 20 mmHg) was recorded. The dp/dt_{max} was reduced at 1062 mm/s, but the cardiac index was normal at 6,4 l/min/m^2. LV cine angiography delineated antero-apical hypokinesia and mild mitral incompetence. There was extensive calcifi-

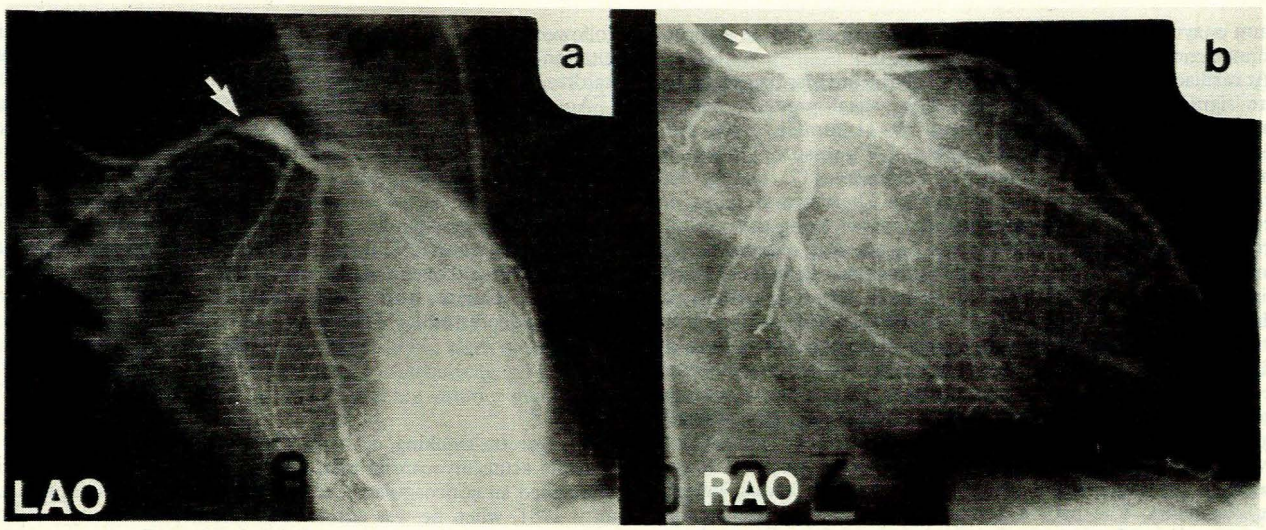

Fig. 1. Case 1. Left coronary cine angiograms in (a) shallow left anterior oblique (LAO), and (b) right anterior oblique (RAO) projections. A subtotal ostial stenosis of the left mainstem coronary artery is visualized (arrowed). **There is also extensive left-to-left collateral blood flow.**

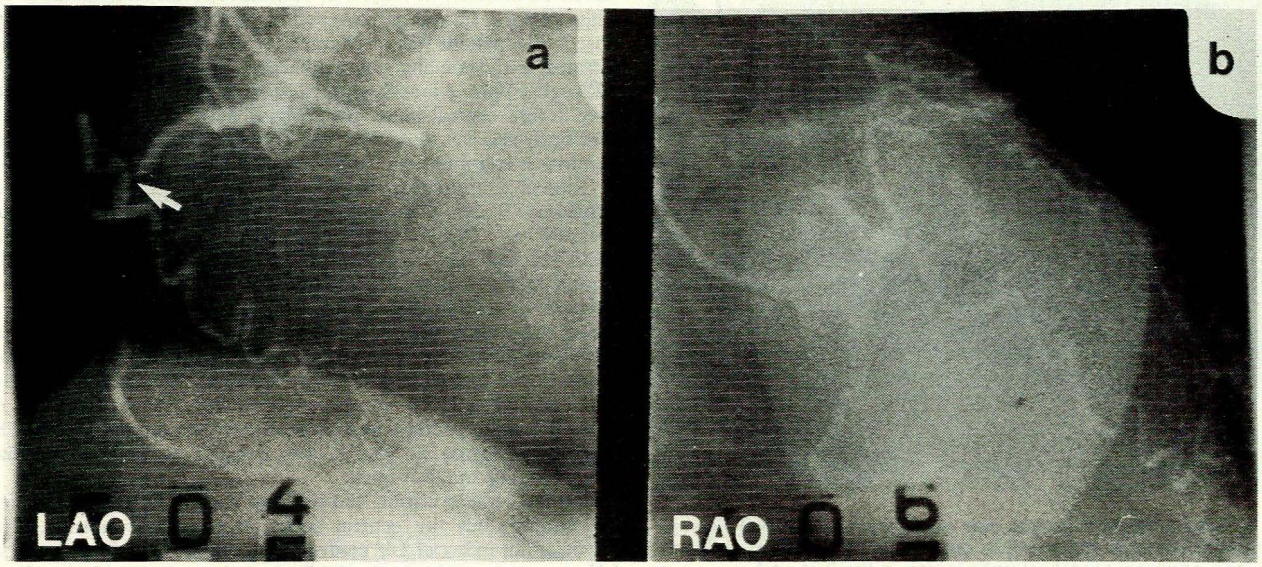

Fig. 2. Case 2. Right coronary cine angiograms in (a) LAO and (b) RAO views. This dominant vessel is diffusely diseased and has a total occlusion (arrowed) **in its first part. Right-to-right, as well as right-to-left collateralization are in evidence.**

cation of the proximal parts of the coronary arteries. The RCA was dominant, diffusely diseased, and totally occluded in its first part, the distal vessel being reconstituted by right-to-right collateral flow (Fig. 2). Some retrograde filling of the LCA by way of right-to-left collateral vessels was also visualized. The LCA cine angiograms delineated a subtotal obstruction of the ostium with diffuse disease of the remaining vasculature.

Within minutes of completion of the last coronary cine angiogram the patient complained of very severe central chest pain, followed by marked sinus bradycardia, vomiting and hypotension. Resuscitation measures, including temporary transvenous cardiac pacing, were instituted. Repeated episodes of ventricular fibrillation necessitated electrical defibrillation. Resuscitation proved unsuccessful. Autopsy was not performed. The cause of death is believed to have been an acute anterior myocardial infarction.

Case 3

This 56-year-old white woman was asymptomatic until 1 week before being seen at the Cardiac Clinic at Tygerberg Hospital. She gave a history of effort-related angina pectoris which within days became far more severe and occurred at rest. Her general practitioner began therapy with nitrates, β-blockers and calcium antagonists, but this proved ineffective. The patient had no risk factors for IHD.

On admission to the ICCU at Tygerberg Hospital on 27 May 1983 she appeared healthy. Her blood pressure was 130/80 mmHg and her pulse rate 56/min, with no signs of cardiac failure. There was no cardiomegaly but there were signs of possible aortic valvular stenosis. A resting ECG demonstrated sinus rhythm of 60/min, a P-R interval of 0,16 second, and mean QRS axis of +60°. There were no features of LV or atrial enlargement. However, 2 mm downward-sloping ST-segment depression was seen in the anterolateral and anteroseptal leads, and the inferior leads had 1 mm downward-sloping ST-segment depression. A chest radiograph showed a normal cardiac silhouette, some unfolding of the aortic arch, no aortic valve calcification and clear lung fields. In view of the possibility of aortic stenosis, echocardiography was attempted, but was unsuccessful. The patient was initially unwilling to undergo cardiac catheterization, but since her chest pain remained relentless she finally asked for it to be done with a view to possible emergency coronary artery bypass. Serial

serum enzyme investigations remained normal and acute myocardial infarction was excluded.

At cardiac catheterization on 2 June, all the intracardiac pressures were significantly elevated. Mean right atrial pressure was 18 mmHg, right ventricular pressure 64/9-11 mmHg, main pulmonary artery pressure 64/35 mmHg, LV pressure 178/24-36 mmHg, central aortic pressure 178/68 (mean 112) mmHg and mean capillary wedge pressure 36 mmHg. There was therefore no aortic stenosis and the murmur was accepted as being the result of aortic nodular sclerosis. The cardiac output was reduced at 3,2 l/min. LV cine angiography (RAO projection) delineated generalized hypokinesia with trivial presystolic mitral insufficiency. Selective coronary arteriography demonstrated a 95% ostial stenosis of the RCA with an additional 70% long-segment lesion in its midportion (Fig. 3). The LCA displayed a 95% tapering stenosis of the left mainstem ostium. In addition, there was a subtotal obstruction of the origin of the left circumflex (lcx) coronary artery (Fig. 4). No coronary artery calcification or collateral blood flow could be seen. Severe proximal triple-vessel coronary artery disease, complicated by significant biventricular cardiac failure, was diagnosed. The patient had no angina pectoris during the procedure but 15 minutes after completion of cardiac catheterization she suddenly developed acute pulmonary oedema, rapidly followed by marked sinus bradycardia and asystole. Routine resuscitation measures including insertion of a temporary transvenous cardiac pacemaker were unsuccessful.

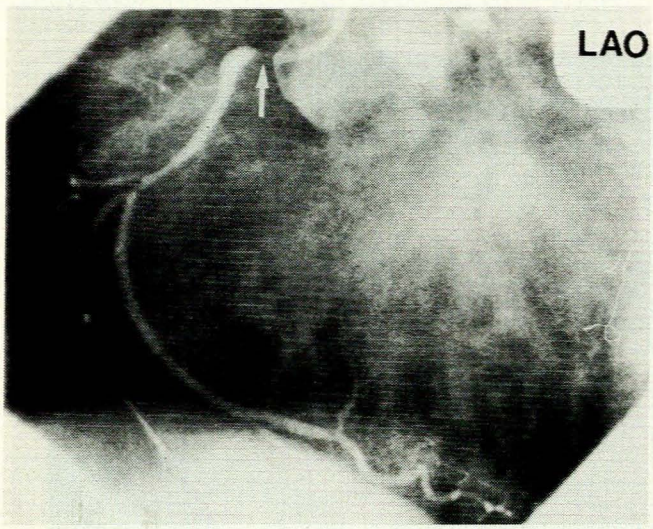

Fig. 3. Case 3. Right coronary cine angiogram in the LAO view. A 95% ostial stenosis (arrowed) **is clearly evident. There is also a 70% long-segment atherosclerotic lesion in the first part.**

Autopsy demonstrated virtually total occlusion of both coronary ostia by markedly advanced atherosclerosis which had extended into the proximal parts of all major vessels. There were no features of possible iatrogenic coronary artery dissection. The myocardium showed evidence of an extensive and acute anterior myocardial infarction. Examination of the other viscera confirmed the presence of congestive cardiac failure. The cause of death was thus considered to be cardiogenic shock, precipitated by cardiac catheterization, in a patient with severe bi-ostial coronary stenosis and markedly depressed LV function.

Case 4

A cigarette-smoking 53-year-old white man had suffered a possible acute myocardial infarction in 1972, after which he was prescribed an anticoagulant and nifedipine. He was asymptomatic until 1981, when he was admitted to another university hospital with an acute inferior myocardial infarction. Coronary arteriography was recommended but the patient refused it. Approximately 3 years later, on 3 June 1983, he was admitted to the ICCU at Tygerberg Hospital with a 6-week history of increasing angina which culminated in angina at rest, despite medical therapy. An acute non-transmural anteroseptal myocardial infarction was diagnosed. The patient was discharged on 17 June 1983 on nifedipine, nitrates, a diuretic and digoxin because he was in LV failure. On 19 August 1983, 2 months after discharge, he was readmitted with very severe central chest pain which had begun some 5 hours previously and was associated with nausea and dyspnoea, unrelieved by nitrates and nifedipine and needing opiates.

Clinical examination revealed no features of hyperlipoproteinaemia. There were signs of mild left heart failure; blood pressure was 160/90 mmHg and pulse rate 86/min. A resting ECG revealed sinus rhythm of 73/min, a P-R interval of 0,13 second and a mean QRS axis of +59°. There were numerous unifocal ventricular extrasystoles, left atrial enlargement and evidence of an old transmural inferior myocardial infarction. There was also poor R-wave progression over the anteroseptal leads and 1 mm downward-sloping ST-segment depression anterolaterally. Serial serum enzyme values were normal and a diagnosis of unstable angina pectoris was made. Since the patient continued to experience angina at rest it was decided to perform cardiac catheterization on 24 August.

Cardiac catheterization showed that the LVEDP was elevated (16 mmHg) but the central aortic pressure was normal. The dp/dt_{max} (an index of LV contractility) was normal (2 300 mm/s). LV cine angiography in the RAO view showed severe generalized

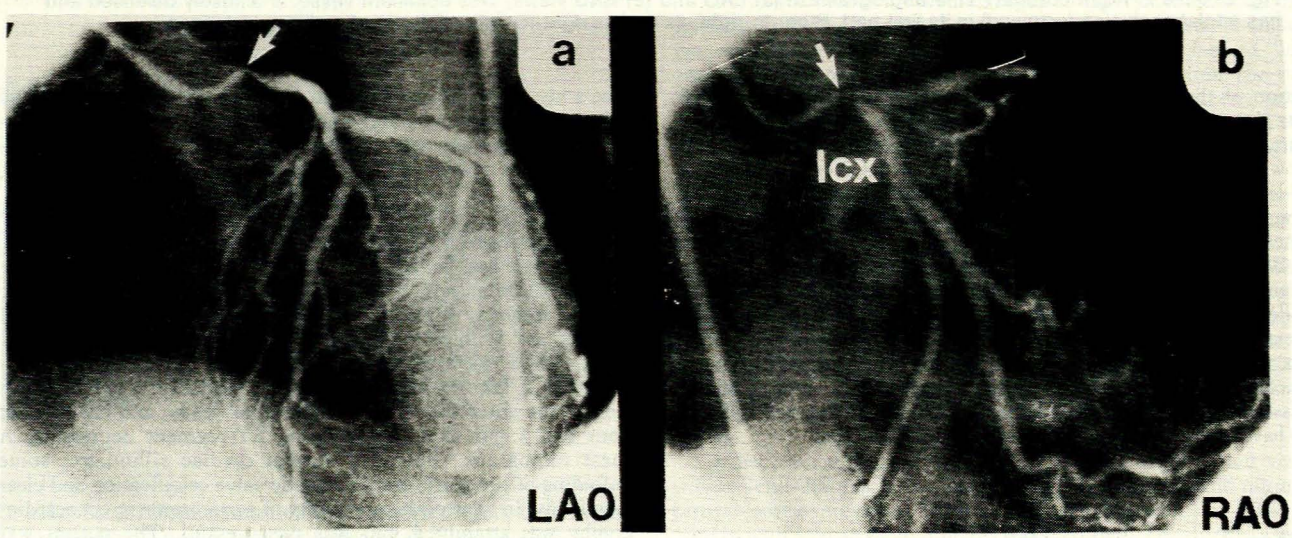

Fig. 4. Case 3. Left coronary cine angiograms in (a) shallow LAO projection with cranial angulation, and (b) shallow RAO view with caudal angulation. A subtotal ostial stenosis (arrowed) **of the left mainstem coronary artery is present. The origin of the lcx coronary artery also has a subtotal obstruction.**

hypokinesia in the absence of any mitral insufficiency. Selective coronary angiography delineated a subtotal obstruction of the RCA ostium and diffuse disease of this vessel (Fig. 5). The LCA had a 95% ostial stenosis with extension of the atherosclerotic disease process into the proximal aspects of both the left anterior descending (LAD) and lcx coronary arteries themselves fairly diffusely diseased (Fig. 6). There was no evidence of coronary calcification or significant collateral vessel formation. Within minutes of completion of coronary arteriography the patient complained of very severe central chest pain; this was unrelieved by sublingual nitrate and nifedipine. Ventricular fibrillation then appeared which was successfully converted to a sinus bradycardia, but this soon degenerated into ventricular standstill. A temporary transvenous cardiac pacemaker was inserted but electromechanical dissociation occurred and resuscitation was abandoned. The cause of death was considered to be a massive acute anterior myocardial infarction.

Case 5

This patient was a 45-year-old white woman with type II hyperlipoproteinaemia and a family history of IHD and death at an early age. Despite this she smoked heavily. She was totally asymptomatic until 3 weeks before referral to Tygerberg Hospital for cardiac catheterization, when classic effort-induced angina pectoris began, occurring also at rest during the last week at home. An effort test while she was on anti-anginal medication had demonstrated 2 mm horizontal ST-segment depression in leads V3 - V6, and 1 mm horizontal ST-segment depression in leads I and aVL. These ischaemic changes were accompanied by angina pectoris. The patient was admitted to the Cardiac Unit at Tygerberg Hospital on 28 November 1984 for coronary arteriography. She was slightly overweight with the florid features of hyperlipoproteinaemia. Blood pressure was 120/80 mmHg, there was no cardiomegaly but a loud 4th heart sound, and no signs of cardiac failure. A resting ECG was normal with sinus rhythm of 62/min, a P-R interval of 0,14 second and a mean QRS axis of +40°. The chest radiograph was normal.

Cardiac catheterization was carried out on 29 November, and revealed an elevated LVEDP of 19 mmHg and a normal central aortic pressure, with dp/dt_{max} normal at 2073 mm/s. LV cine angiography delineated slight segmental anterior wall hypokinesia and a normal mitral valve apparatus. Selective coronary angiography showed a normal and non-dominant RCA with no right-to-left coronary collateral flow. Injection of contrast medium into

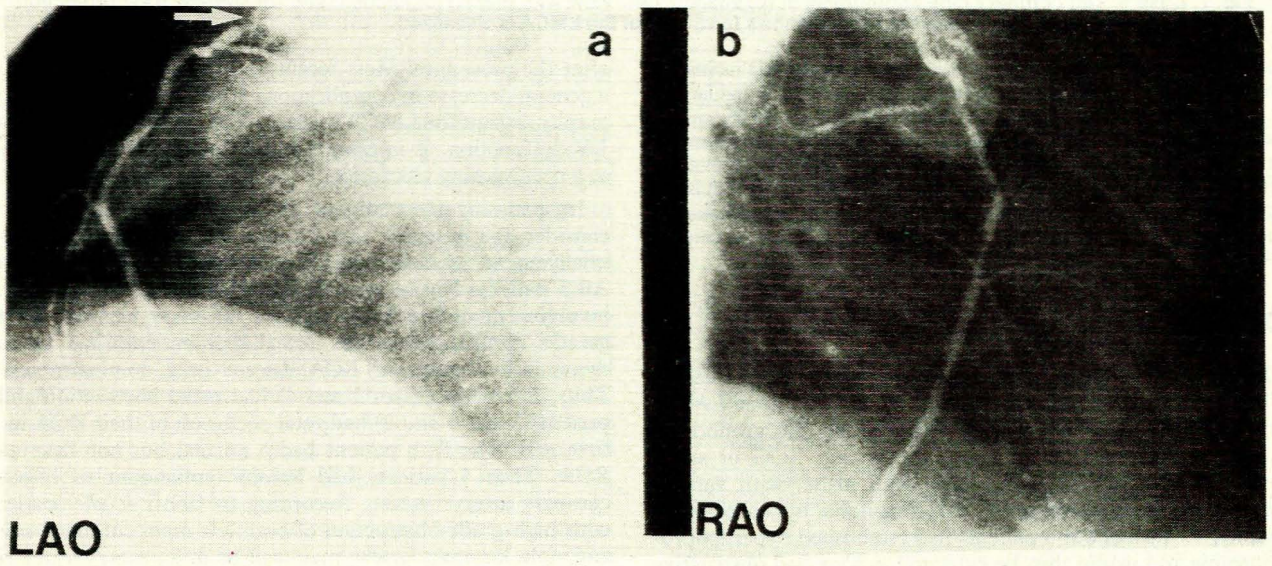

Fig. 5. Case 4. Right coronary cine angiograms in (a) LAO and (b) RAO projections. A subtotal ostial obstruction is present (arrowed) and the vessel is diffusely diseased.

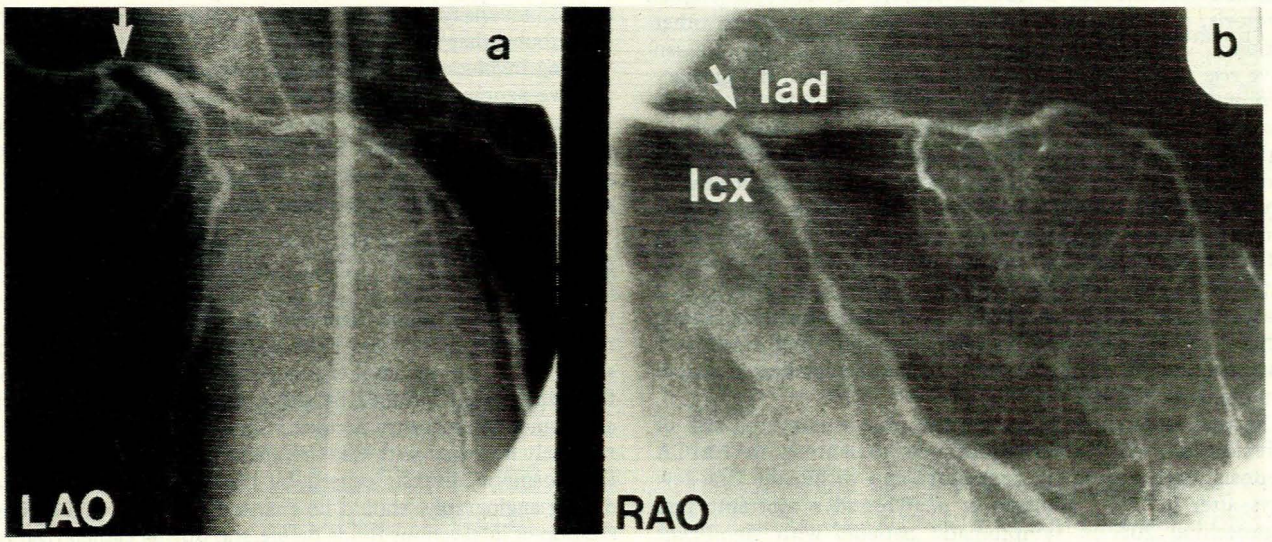

Fig. 6. Case 4. Left coronary cine angiograms in (a) shallow LAO projection with cranial angulation, and (b) shallow RAO view with caudal angulation. A 95% ostial stenosis (arrowed) of the LMCA is in evidence. Furthermore, there is significant involvement of the origins of both the left anterior descending and lcx coronary arteries.

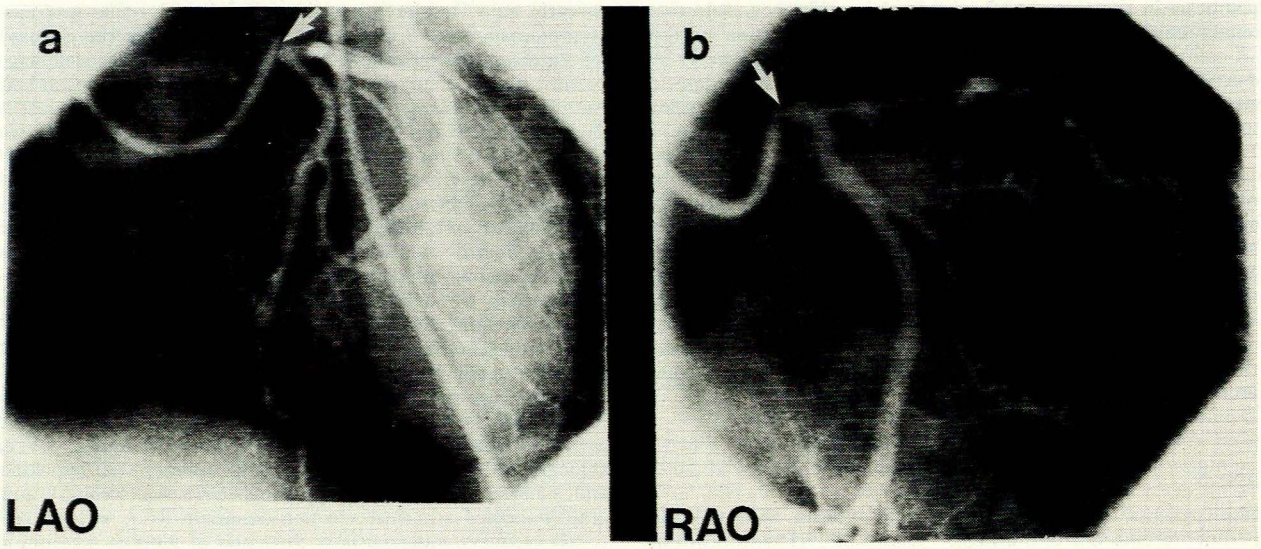

Fig. 7. Case 5. Left coronary cine angiograms in (a) shallow LAO view with cranial angulation, and (b) shallow RAO projection with caudal angulation. A very severe ostial stenosis (arrowed) **of the LMCA is visualized.**

the LCA demonstrated a severe ostial stenosis of the left mainstem coronary artery (LMCA) with minor internal luminal irregularities in the other vessels (Fig. 7). There was no coronary artery calcification. Soon after the last contrast injection central aortic pressure fell and sinus bradycardia rapidly set in. All resuscitation attempts failed. The cause of death was considered to be a massive acute anterior myocardial infarction due to sudden occlusion of the severely obstructed LMCA in the presence of a non-dominant RCA.

Discussion

The LMCA has been termed 'the artery of sudden death'.[1] This definition has been supported by the fact that LMCA involvement cannot be reliably excluded on the grounds of symptoms, physical examination, the resting ECG or stress testing.[2–7] Recently attempts have been made, with varying degrees of success, to make this diagnosis by echocardiography.[8–13] Two of our 5 patients had established hyperlipoproteinaemia and would thus be expected to have had obstructive coronary atherosclerosis. Moreover, all were aged 45 years or older at the time of coronary arteriography and the majority smoked heavily. All 5 gave a history of unstable angina pectoris, a finding reported in 17 - 80% of patients in other studies.[2,14,15] Gulotta[16] has reported that LMCA stenosis can give rise to a higher frequency of dyspnoea than the primary symptom, probably due to cardiac failure secondary to a large mass of ischaemic myocardium. However, this has not been substantiated by other researchers.[3,6,17] Przybojewski and Rossouw[18] recently documented the case of a young man with severe isolated LMCA obstruction and recurrent acute pulmonary oedema associated with angina pectoris. The resting ECG in our 5 patients was not really helpful in the non-invasive diagnosis of LMCA disease, results being normal in 2 (cases 1 and 5), with nonspecific ST-T-wave alterations in the other 3. Stress electrocardiography was only performed in 1 patient (case 5), since the other 4 had demonstrated severe symptoms in hospital.

Several researchers have reported on the increased risk of selective coronary arteriography in patients with LMCA stenosis, both during the procedure and within the first few days after it.[5–7,19–24] Wolfson *et al.*[24] found a mortality rate approaching 20% in symptomatic patients with significant LMCA obstruction, 3 of their 4 patients dying from hypotension while still on the catheterization table, and the remaining patient dying of irreversible ventricular tachycardia 4 hours after the procedure. Most recently, Gwost *et al.*[25] reported on a general decrease in complications related to coronary arteriography, except for LMCA stenosis. The presence of significant LV dysfunction, as in cases 3 and 4, has also been established as a predisposing risk factor.[26–28]

In analysing the cause of death, it is most important to consider the severity and distribution of coronary arterial involvement, as determined angiographically in our 5 cases. All 5 patients had severe LMCA ostial stenosis with varying involvement of the LAD and lcx coronary arteries. Of far greater significance is the fact that 4 patients had haemodynamically important RCA disease in a dominant vessel. Thus, 2 patients (cases 3 and 4) had ostial obstruction and 2 patients (cases 1 and 2) had total occlusion of their RCA in its first part. The fifth patient had a normal but non-dominant RCA. Thus 4 patients had lesions tantamount to bi-ostial coronary artery disease. According to Conti *et al.*[3] 'patients with high-grade obstruction of both left main coronary artery and right coronary artery may well be at an even greater risk than the majority of patients with LMCAS [left main coronary artery stenosis]'. They noted that LMCA ostial lesions in patients with LMCA disease, although quite rare (7,5% of cases), adversely influenced the prognosis. Most researchers have found that LMCA stenosis is invariably accompanied by varying involvement of the other coronary arteries.[3,5–7,21] The relative paucity of coronary collateral formation in our 5 cases has an important bearing on the risk of the catheterization procedure, since critical ischaemia can ensue with contrast injection. The presence of these coronary collaterals is vital in patients with total LMCA obstruction.[29,30]

On account of the greater risks of selective coronary arteriography in patients with LMCA stenosis, especially if there is associated significant RCA obstruction and depressed LV function, several researchers have recommended certain technical precautions.[3] It has been suggested that these patients should all be on maximal anti-anginal medication but without the hypotension induced by these drugs. This is usually the case, since the majority present with unstable angina pectoris, routinely managed with this therapeutic regimen. Dehydration from osmotic diuresis caused by the contrast medium used during angiography should be guarded against by the administration of intravenous fluids before and during catheterization. Great care should be exercised in ensuring that marked pressure decrease with engagement of the coronary ostium does not occur. In order to avoid this, 'cusp injection' or non-selective

angiography is advised, as well as the minimum number of contrast injections with adequate pauses in between to ensure minimal iatrogenic myocardial ischaemia. Iskandrian *et al.*[17] strongly advise prompt administration of nitroglycerin if the patient experiences angina pectoris. Wolfson *et al.*[24] comment that 'these patients are high priority candidates for coronary bypass surgery as well as high risk candidates for coronary arteriography', and that the 'definition of the precise coronary anatomic deficit is essential in these patients, and those at the greatest risk need the study most urgently'. However, they have reservations about non-selective coronary arteriography because 'in many cases, this approach yields non-diagnostic studies, inadequate for decisions influencing surgical therapy'.

During the last few years, the employment of intra-aortic balloon counterpulsation (IABC) in patients with LMCA stenosis has been recommended both during the cardiac catheterization procedure and during the subsequent coronary artery bypass operation.[31-33] However, the complications of IABC itself must be borne in mind. It cannot be stressed too strongly that once the diagnosis of significant operable LMCA disease has been established the patients must undergo surgery within hours of completion of cardiac catheterization since they are definitely at high risk for complications. World experience has documented the superiority of surgery in these patients, who should therefore not continue on medical therapy.[34,35]

The sequence of events immediately after selective coronary arteriography in our 5 patients indicates a uniform pattern. Severe chest pain was usually followed by hypotension and electromechanical dissociation unresponsive to emergency interventions, including insertion of a temporary transvenous right ventricular pacemaker. This picture is one of global myocardial ischaemia. The possible superimposition of vasospasm on the fixed LMCA stenosis must be seriously considered. Catheter-induced spasm, especially of the RCA, has been extensively documented in the literature. Vasospasm of the LMCA has been described most infrequently.[36,37] Iatrogenic dissection of the LMCA is also possible, and this likelihood may well be lessened by the use of a recently-introduced Softip cardiovascular catheter (Angiomedics Incorporated, Minneapolis).[38,39] However, it must be appreciated that significant LMCA ostial stenosis associated with haemodynamically important RCA disease, in the absence of extensive coronary collateral flow and in the presence of reduced LV contractility, puts the patient at extremely high risk of death during or immediately after diagnostic selective coronary arteriography. Since this combination is relatively common in a population with a high incidence of coronary atherosclerosis, particularly with underlying hypercholesterolaemia, it is important for a safe method of coronary artery definition to be found.

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