Reversible hypovolaemic shock and myocardial ischaemia caused by contrast medium administered during diagnostic cardiac angiography

A case report

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Summary

A 65-year-old white man with severe symptomatic four-vessel atherosclerotic coronary artery disease underwent selective coronary arteriography. Two hours after this procedure he developed hypovolaemic shock secondary to the hyperosmolar contrast medium, as well as severe angina pectoris accompanied by myocardial ischaemia. This diagnosis was established with the aid of Swan-Ganz catheterisation and the patient was successfully managed with intravenous fluid replacement and emergency coronary artery bypass graft surgery. Pathophysiological aspects are discussed with comments on the possible prevention of such a potentially life-threatening complication of selective coronary angiography.

Case report

The patient was a 65-year-old white man who had been a heavy cigarette smoker until 7 years previously. His essential hypertension had been controlled by a β-blocker, atenolol. A younger brother had significant ischaemic heart disease (IHD). Classic effort-induced angina pectoris was first noted 2 months before his admission to the Cardiac Unit, Tygerberg Hospital, on 8 January 1985 for further investigation. His general practitioner had prescribed diltaizem, a transdermal nitroglycerine preparation and diltiazem, a transdermal nitroglycerine preparation and with emotion and he was referred for coronary bypass surgery. Pathophysiological aspects are discussed with comments on the possible prevention of such a potentially life-threatening complication of selective coronary angiography.

Cardiac catheterisation

Central aortic and LV pressures were within the normal range. LV cine angiography (right anterior oblique (RAO) projection) demonstrated a hypercontractile and markedly hypertrophic chamber, the angiographic features suggesting the possibility of HOCM (Fig. 1). There was no evidence of mitral-valve prolapse or insufficiency. The right coronary artery (RCA) was studied in both the left anterior oblique (LAO) and RAO view. The RCA was seen to be a dominant and ectatic vessel with three significant stenoses in the first half (Fig. 2). No right-to-left collateral blood flow could be seen, the aortic pressure was stable, and the patient did not complain of angina. The left coronary artery (LCA) was injected in four different views (steep LAO, shallow LAO with cranial angulation, shallow RAO with caudal tilt), each injection consisting of a bolus of approximately 8 ml of Urographin-76%. Adequate time was allowed between injections for stabilisation of the aortic pressure (normal with catheter engagement of the LCA ostium). There was no angiogram and there were no cardiac arrhythmias. The cine angiograms delineated two subtotal obstructions in the mid-portion of the LCA, the distal one just proximal to the origin of the left anterior descending (lad) and left circumflex (lc) coronary arteries (Figs 3 and 4). Further significant stenoses were seen in the lad coronary artery distal to the origin of its first diagonal branch, and in the lc coronary artery proximal to its mid-lateral branch (Figs 3 and 4). The cardiac catheterisation was completed within 30 minutes and the patient was haemodynamically stable with no angina pectoris.

Course after cardiac catheterisation

In view of the severity of the coronary artery disease, especially the LMCA stenoses, as well as the fact that the vessels were operable, emergency coronary artery bypass graft (CABG) surgery

Cardiac catheterisation
was discussed and arrangements were made for the patient to return to the Cardiac Unit for monitoring until such time as the theatre could be prepared. The patient was also instructed to take oral fluids freely. On returning to the Cardiac Unit a resting 12-lead ECG was unchanged from that before cardiac catheterisation. About 2 hours later the patient complained of sudden severe precordial pain. A resting ECG was immediately recorded and demonstrated 3 mm downward sloping ST-segment depression in the anterolateral leads. The patient was also found to be shocked with a blood pressure of 75/20 mmHg and a pulse rate of 64/min. An intravenous line was immediately set up and Plasmalyte-B was slowly infused, together with the inotrope, dobutamine. A Swan-Ganz catheter was inserted percutaneously via the left subclavian vein and the right heart pressures recorded. Main pulmonary artery pressure was normal at 22/5 (mean 12) mmHg with a low mean pulmonary capillary wedge pressure (PCWP) of 4 mmHg. A diagnosis of intravascular fluid depletion, probably caused by the osmotic effect of the contrast medium, precipitating myocardial ischaemia, was made; acute myocardial infarction was also a possibility.

The patient received 3 litres of Plasmalyte-B intravenously within 1 hour with continuous haemodynamic monitoring. Inotropic drug therapy was discontinued; fluid replacement raised the blood pressure to 130/80 mmHg and the mean PCWP to 8 mmHg. All the signs of shock had now disappeared, and the patient began to pass a small volume of concentrated urine. His chest pain also disappeared and the ECG now demonstrated resolution of the previous myocardial ischaemia and no signs of acute myocardial infarction. Intravenous fluid was continuously infused with the PCWP as a guide to volume required. In this stable clinical condition the patient was taken to the operating theatre where his cardiac output was recorded as 6,8 l/min, cardiac index 3,9 l/min/m², blood pressure 130/80 mmHg, mean PCWP 13 mmHg and pulmonary artery pressure 21/6 (mean 12) mmHg. Emergency CABGs were then successfully inserted into the posterior descending branch of the RCA, the lad
LAO

Fig. 3. Left coronary cine angiograms in (a) steep LAO view; and (b) shallow LAO projection with cranial angulation. Two subtotal obstructions (arrowed) are seen in the mid-portion of the left mainstem coronary artery (b). There is also a significant obstructive lesion (arrowed) in the left anterior descending (lad) and left circumflex (lc) coronary artery, the latter not being clearly evident in these projections.

Fig. 4. Left coronary cine angiograms in (a) shallow RAO with cranial angulation; and (b) shallow RAO with caudal angulation. Subtotal obstructions (arrowed) are visible in both the lad and lc coronary arteries. One of the stenoses (arrowed) in the left mainstem coronary artery is evident in (b).

coronary artery both proximal and distal to the stenosis, and the anterolateral branch of the lc coronary artery.
The patient made an uneventful recovery and was discharged on 22 January 1985 on atenolol 100 mg daily, prazocin 2 mg 3 times daily, dipyridamole 100 mg 3 times daily and aspirin 300 mg daily. Follow-up documented the absence of any angina pectoris and adequately controlled essential hypertension. Repeat treadmill stress testing was negative.

Discussion

This experience highlights several important aspects of the management of symptomatic atheromatous LMCA disease. The concomitant presence of significant narrowing of the RCA, as well as severe symmetrical LV hypertrophy secondary to chronic essential hypertension, had an important influence on the clinical picture.

Several authors have attempted to define the clinical characteristics of patients with LMCA involvement with a view to timely coronary arteriography and possibly urgent surgical intervention. Unfortunately, no clinical picture pathognomonic of underlying LMCA obstruction has been established, although marked ST-segment depression on stress ECG, as seen in our patient, is suggestive. Intermittent pulmonary oedema secondary to global myocardial ischaemia has also recently been encountered in such cases. The early
diagnosis of LMCA disease is important since CABG surgery
significantly improves the prognosis.

Selective coronary arteriography still remains the 'gold-
standard' diagnostic tool, but is known to be associated with a
greater risk.4-6,10-15 Recently, Przybojewski16 documented
the death during cardiac catheterisation of 5 patients with LMCA
ostial stenosis over a period of 10 years. Mortality related to
coronary arteriography in patients with LMCA obstruction
has been reported by Wolfsen et al.15 to be as high as 20%,
but most recently Kron17 commented on a decrease in frequency
in this subgroup of patients. Intravascular dehydration, second-
dary to the hyperviscous effect of the contrast medium used
during arteriography, may be a precipitating factor for
increased risk in cases of LMCA disease. Researchers therefore
recommend a minimum of contrast injections and administra-
tion of intravenous fluid during the procedure.

Haemodynamic assessment by Swan-Ganz catheterisation 2 hours after successful completion of coronary arteriography in
our patient established dehydration as the mechanism for
hypovolaemic shock. This was accompanied by severe angina
pectoris and objective myocardial ischaemia as demonstrated on
the resting ECG. Intravenous fluid administration rapidly
reversed this shock.

Recently Coetzee et al.18 showed experimental evidence for
a hypotension-induced fall in coronary perfusion pressure
(CPP) giving rise to severe regional myocardial dysfunction in
the presence of fixed critical coronary artery obstruction, and
commented that 'it appears to be of primary importance to
maintain normal blood pressure in patients with ischaemic
heart disease, and it may even be preferable to increase the
blood pressure slightly during the peri-operative period'. These
findings are of direct relevance to our case, since the calculated
CPP was 33 mmHg, a figure significantly below normal, con-
sidering that effective autoregulation takes place when the
CPP ranges from 50 mmHg to 150 mmHg in the absence of
coronary artery obstruction.19 In the presence of significant
coronary artery stenosis there is maximum autoregulation, as
represented by maximal dilatation of the artery segment distal
to the stenosis; under these circumstances coronary blood flow
will depend solely upon the perfusion or driving pressure.

Other important factors have to be considered. Hoffman20
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LV hypertrophy, can contribute to subendocardial ischaemia.
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