Unstable angina pectoris secondary to multiple calcified coronary artery masses

Successful treatment with coronary artery bypass surgery

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Summary

A 31-year-old doctor's wife suffered from severe unstable angina pectoris (AP) due to two large, heavily calcified masses involving the right coronary artery and the left anterior descending branch of the left coronary artery. The causes of the masses could not be determined with certainty, but in view of the history (which included the ingestion of large quantities of raw *boerewors* (traditional spiced sausage) and histopathological findings, we believe that they were coronary artery aneurysms which developed secondary to coronary arteritis many years previously. The possibility of echinococcal (hydatid) infection is also discussed. Cardiac surgery entailed total excision of both masses, together with sections of their accompanying coronary arteries which had become fibrotic as a result of the arteritis, and re-establishment of coronary blood flow by the insertion of two saphenous vein coronary artery bypass grafts. Her AP was dramatically relieved and she continues to be asymptomatic without taking anti-anginal drugs.

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Case report

The 31-year-old wife of a colleague who practised in a sheep-rearing area of the Cape Province had never smoked and had no risk factors for ischaemic heart disease (IHD). Some 5 years before admission and during her last pregnancy she took a fancy to eating large quantities of raw *boerewors* (a traditional spiced sausage). This eating habit continued and was of some concern to her husband.

Approximately 20 years previously she had contracted a severe generalized illness with high fever, swollen neck glands, diffuse arthralgia and a skin rash. She remembers that a diagnosis of 'glandular fever' was made and that she was hospitalized for several weeks. Four years previously she complained of tiredness, nonspecific chest pain, headaches and abdominal discomfort. For the last 3 years she suffered from diffuse arthralgia, particularly of the distal interphalangeal and temporomandibular joints, without signs of arthritis; she also noted intermittent painful and raised nodules over the anterior aspect of both tibiae, but the presence of these was never substantiated by a dermatologist. Two weeks before admission to Tygerberg Hospital she noted a sensory disturbance over the left side of her face but no abnormality was found and this symptom cleared within a few days.

Careful interrogation elicited a history of classic effort-induced angina pectoris (AP) during the preceding 18 months. These attacks had become far more frequent during the 4 weeks before admission and had now begun to occur at rest. A transdermal nitrate preparation gave her such severe headaches that this medication was replaced with nifedipine 10 mg 3 times daily. There was a definite reduction in the frequency and severity of the AP, but unfortunately she began having dizzy spells. A physician carried out a two-step Master's stress test which clearly demonstrated 1 mm horizontal ST-segment depression laterally during effort, not associated with chest pain. She was referred to the Cardiac Clinic at Tygerberg Hospital on 24 October 1984.

She was extremely tense with no abnormal clinical signs on general examination. The blood pressure was 120/90 mmHg and the jugular venous pressure was not raised. There was no cardiomegaly and heart sounds were normal. A resting ECG showed sinus rhythm of 75/min, a P-R interval of 0,16 second, a mean QRS axis of +20°, and nonspecific ST-T-wave flattening in the inferior leads. A chest radiograph demonstrated a 'calcified left hilar node' (an observation later proved to be incorrect). Echocardiography (M-mode and two-dimensional) showed no abnormality.

Nifedipine therapy was withdrawn and a stress ECG repeated. A treadmill exercise test on 30 October 1984 with the patient on no maintenance drug therapy and only taking sublingual nitrates when required documented 1,5 mm upward-sloping ST-segment depression inferolaterally at peak exercise in the absence of any AP. A clinical diagnosis of IHD (almost certainly non-atheromatous) was then made and arrangements were made for coronary arteriography.

Results of other investigations, including hepatic, renal and haematological function tests, were normal; collagen screening and serological tests were all negative. Cardiac catheterization was carried out on 15 November 1984. Immediately on fluoroscopy two egg-shaped masses could be seen to move with the heart. Both the right- and left-sided intracardiac pressures were within normal limits. Haemodynamic indices of left ventricular (LV) function were also normal. LV cine angiography in the right anterior oblique (RAO) and left anterior oblique (LAO) projections demonstrated normal contractility, no mitral valve prolapse, and no mitral insufficiency (Fig. 1), but two well-defined calcified masses were visible near the aortic root in the atrioventricular groove and the anterior aspect of the heart (Fig. 1). Selective coronary angiography was then performed. Injection of dye into the dominant right coronary artery (RCA) in the LAO and RAO projections revealed that the lumen in its first part was totally obliterated and that collateralization had ensued around a calcified oval cyst-like structure approximately 2,5 cm in its greatest diameter (Fig. 2). The remainder of the RCA appeared normal. Right-to-left collateral vessels were also seen to fill the left anterior descending (LAD) coronary artery (Fig. 2), which immediately suggested some obstruction in the proximal part of that vessel. Injection of dye into the left coronary artery (LCA), in both LAO and RAO views, delineated a similar but larger mass (approximately

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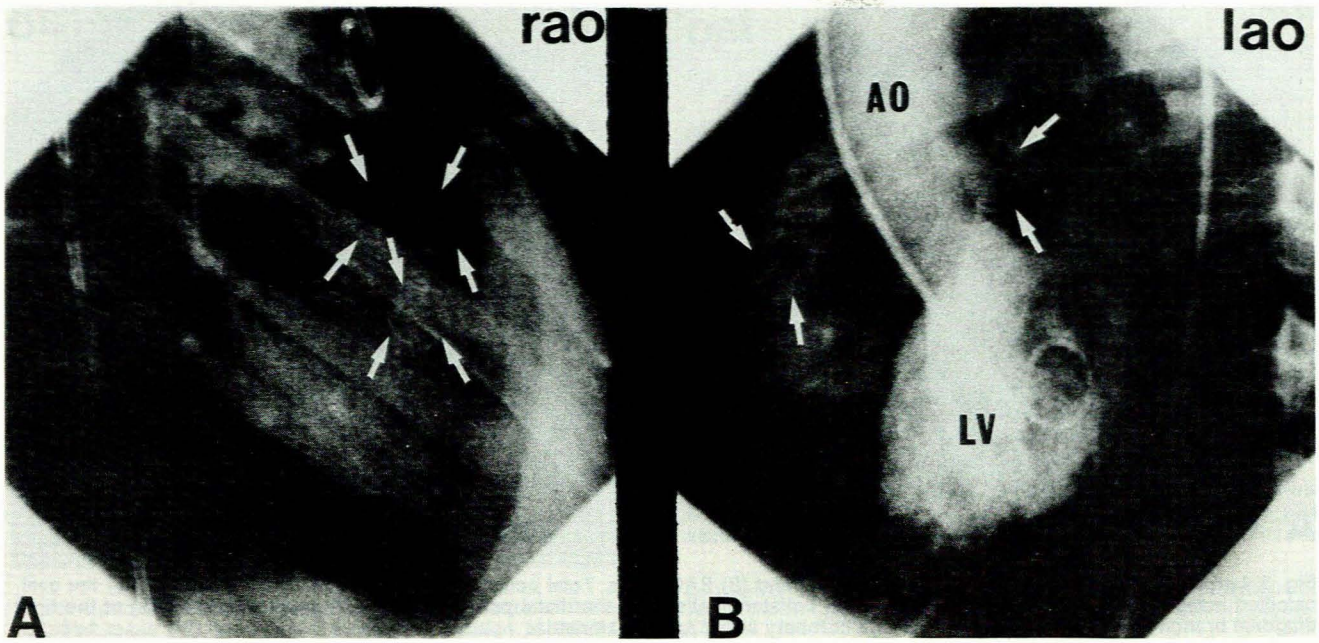

Fig. 1. A — two rings of calcium delineating the masses (arrowed) are visualized in the RAO view; B — LV cine angiogram in the LAO projection showing normal contractility (including that of the interventricular septum). The calcified masses are arrowed. (AO = ascending aorta).

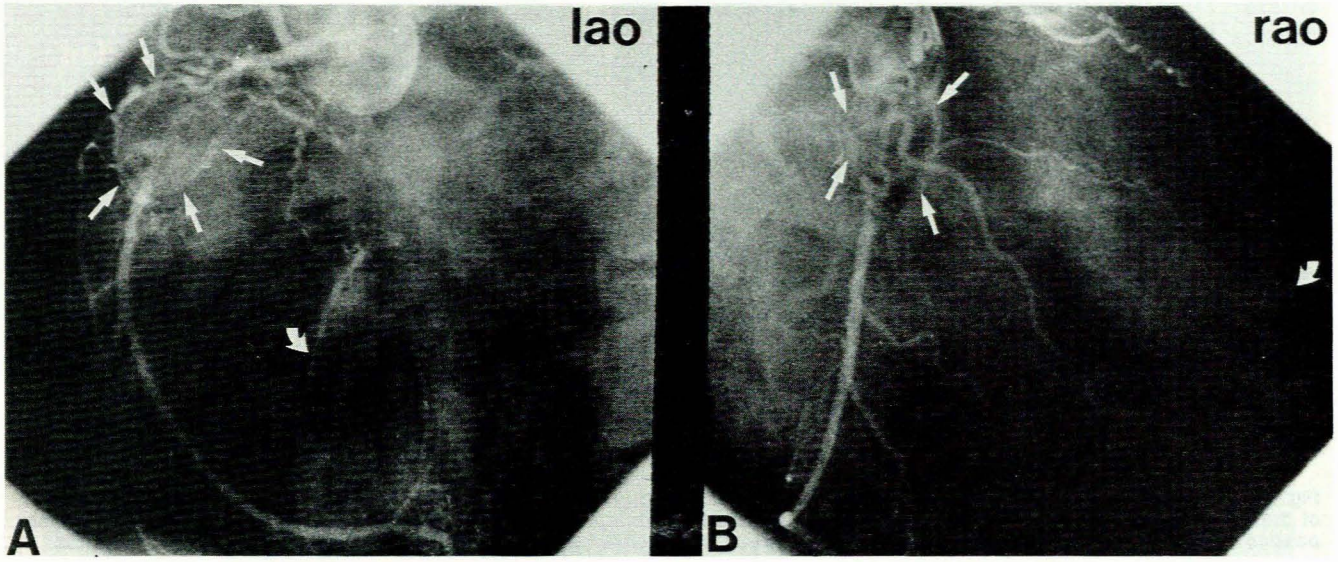

Fig. 2. Right coronary cine angiograms in the (A) LAO and (B) RAO projections. Total occlusion of the proximal part of the coronary artery by the oval calcified mass (arrowed) is seen. Right-to-right collateralization fills the distal portion of the RCA, which appears normal. The LAD branch of the LCA (thick arrows) fills retrogradely from the RCA.

3,5 cm in its longest diameter) which totally occluded the proximal portion of the LAD and was reconstituted by a multitude of left-to-left collaterals, mainly from the left circumflex (LCx) coronary artery (Figs 3 and 4). The LCx itself appeared free of disease on angiography. An aneurysm of the first diagonal branch of the LAD (Figs 3 and 4) was also present. Coronary angiography was performed without the patient experiencing any AP or arrhythmias.

The favoured diagnosis at this juncture, particularly in the light of the patient's history, was that of echinococcal (hydatid) cysts involving the RCA and LAD. Another possibility entertained was that of coronary arteritis with aneurysm formation and subsequent calcification. It was exceptionally difficult to determine angiographically whether the calcified masses occluded the proximal portions of the two coronary arteries by extrinsic pressure, or whether an actual obliterative process had taken place due to direct involvement by the disease. The coronary ostia themselves were entirely normal, as was the ascending aorta, features against a diagnosis of additional aortitis. Echinococcal complement fixation tests were negative, as were collagen tests.

After discussion with the patient's husband and the cardiothoracic surgeons, urgent cardiac surgery was carried out on 19 November 1984, 4 days after the cardiac catheterization.

Examination of the heart revealed heavily calcified and extremely rigid oval masses overlying the proximal course of both the RCA and LAD proximal to the origin of the first diagonal branch (Fig. 5). The former was approximately 2,5 cm in its greatest diameter, whereas the latter was 3,5 cm. The patient was then placed on cardiopulmonary bypass and cooled down to a rectal temperature of 22°C. Cold cardioplegia was used, the heart was made to go into ventricular fibrillation, and the ascending thoracic aorta was cross-clamped. Cold cardioplegic solution was infused into the aorta until total asystole was achieved. An incision was then made

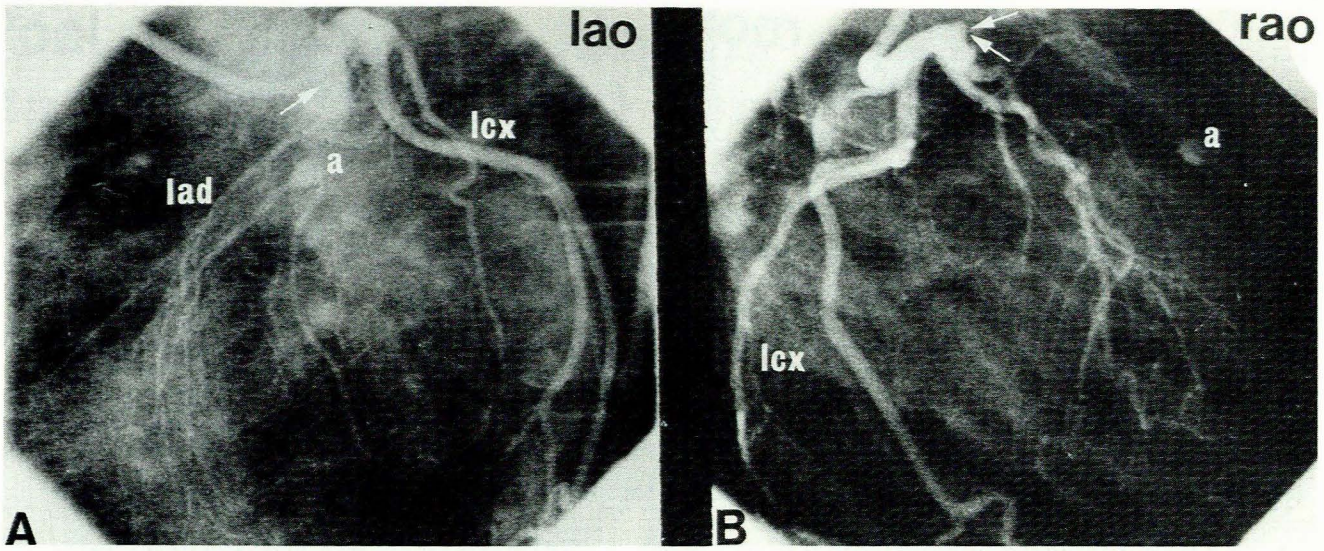

Fig. 3. Left coronary cine angiograms in the (A) LAO and (B) RAO views. Total occlusion of the proximal part of the LAD by the oval calcified mass (arrowed) is visualized. Left-to-left collateralization fills the distal part of the LAD. A small aneurysm (a) of the first diagonal branch of the LAD is present. The LCx coronary artery appears normal.

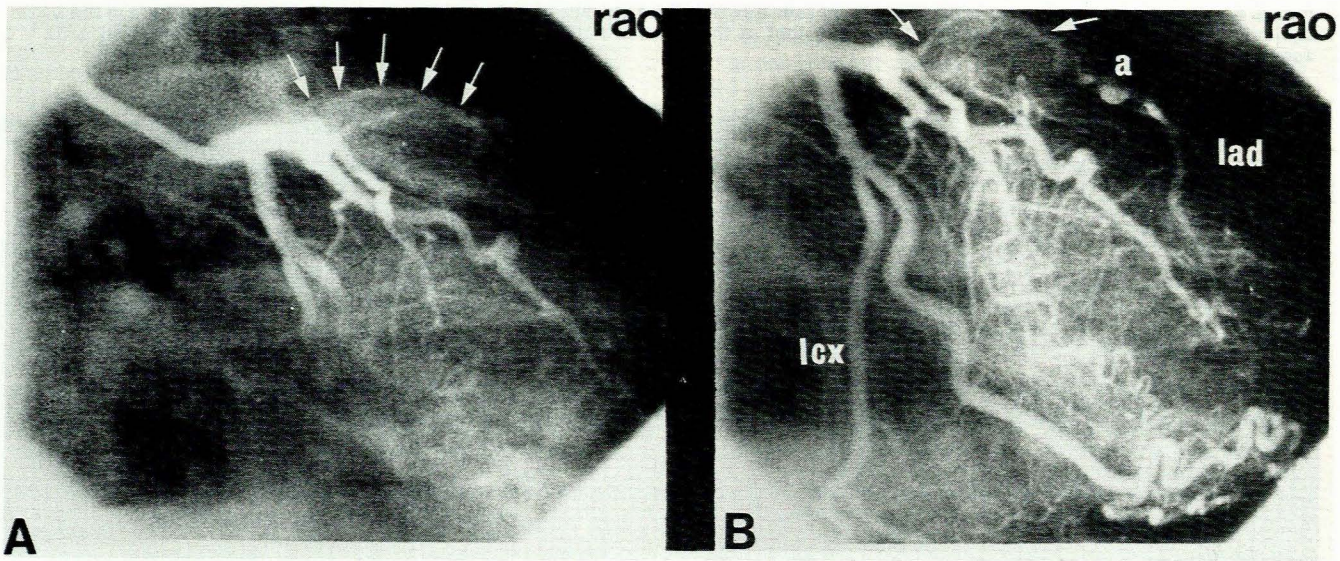

Fig. 4. Left coronary cine angiograms in the RAO projection. The early phase of dye injection (A) clearly demonstrates total occlusion of the LAD by the calcified mass (arrowed). A later phase of angiography (B) shows the extensive collateral network of vessels passing through the interventricular septum. The LCx appears normal and the aneurysm (a) of the first diagonal branch is visualized.

into the mass involving the RCA and this was noted to be 'cystic' and free of any fluid. The mass was then carefully dissected and was found to be connected both proximally and distally to the RCA. The portion of the RCA traversing the mass was totally obliterated and fibrotic. This 'cystic' mass was then totally excised and the two openings of the RCA closed off with 5 - 0 Prolene. The mass involving the LAD, which had similar physical features and relationship to the vessel, was excised and the LAD openings sutured. The LAD was then incised just distal to a very small and soft aneurysm of the first diagonal branch. A 1,75 mm probe was passed into the LAD, which was noted to be patent. A distal saphenous vein anastomosis to the LAD was then carried out with resultant good flow through the coronary artery bypass graft (CABG), as well as retrogradely through the first diagonal branch. The RCA was similarly incised just proximal to the acute angle of the heart. This vessel was seen to be arteriosclerotic but the lumen was patent, allowing a 2 mm diameter probe to pass. The distal anastomosis of the CABG was then made in the RCA with good flow ensuing. The proximal aortic CABG anastomosis of both the LAD and RCA was then completed. After rewarming and weaning off cardiopulmonary bypass, visualization and palpation of both CABGs demonstrated good flow.

While in the operating theatre both 'cysts' were examined macroscopically. It was extremely difficult to bisect these masses due to the thick and dense calcification enveloping them. The interior of both masses consisted of a fairly translucent and white, mucinous, glistening material (Fig. 6). The coronary artery coursing through each mass was seen to be totally obliterated and fibrotic. A large old thrombus was visualized within the mass involving the LAD. Trabeculae of calcified material extended into the interior of both masses.

Both masses had the same histological features and showed a heavily calcified outer portion with fibrous connective tissue of variable density inside, from extremely dense and hyalinized to loose and oedematous tissue. Focal lymphoplasmacytic infiltrates were present as well as a fair number of haemosiderin-laden macrophages. The thrombus in the centre of the mass involving the LAD was confirmed. Although very little residual vessel wall elements could be identified by special stains (Figs 7 - 9), the

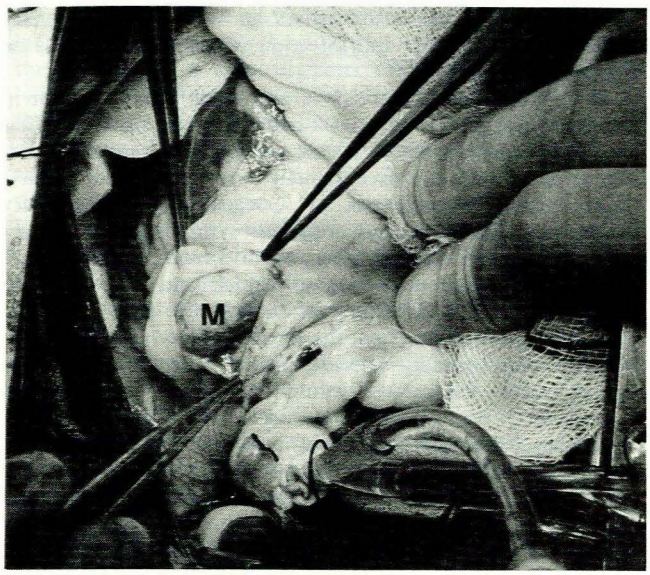

Fig. 5. Operation field demonstrating partial resection of the oval calcified mass (M) involving the proximal portion of the RCA, lying within the atrioventricular groove.

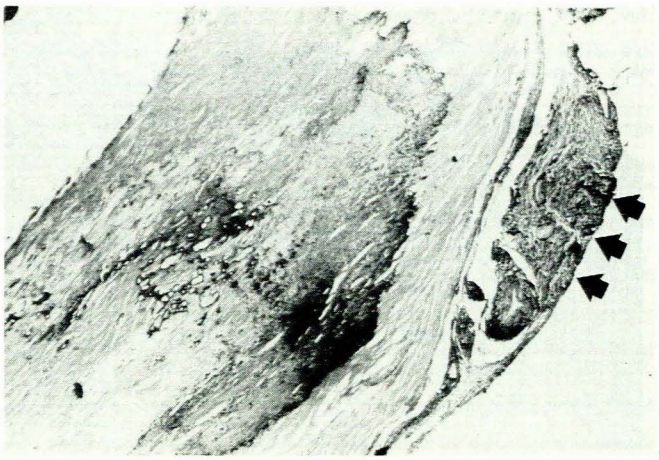

Fig. 7. Section through calcified structure — low-power view showing calcification and dense fibrous connective tissue and vascular wall remnants (arrows) **(Verhoeff x 80).**

histopathological picture could be consistent with heavily calcified aneurysms of the coronary vessels.

The postoperative course was totally uneventful, and the patient was discharged on 24 November 1984, on dipyridamole 100 mg 3 times daily and aspirin 325 mg/d. Follow-up 4 weeks later found her completely free of AP, with a normal resting ECG.

Discussion

Experience with this patient illustrates the great importance of always considering a symptom, in this case chest pain, in a serious light despite the tendency to disregard it in a patient in whom one least expects to uncover a significant cardiac lesion. The 'false-positive' stress ECG in a female patient previously categorized as neurotic is the perfect clinical setting for discouraging further investigation. In retrospect, employment of fluoroscopy would have made for a more confident diagnosis. The observation of a 'calcified left hilar node' on chest radiographs would then have been appreciated to represent one of the calcified cardiac masses.

Selective coronary arteriography established the anatomical cause for the AP. Very proximal obstruction of two major coronary arteries (the RCA and LAD), despite the extensive coronary collateral circulation, was responsible for significant myocardial ischaemia. Fortunately, there was no evidence for previous myocardial infarction (MI) but this complication was highly likely if surgery had not been carried out promptly.

The aetiology of the cardiac lesions is most intriguing. The two calcified masses had their origin in the coronary arteries. Furthermore, the extensive and thick calcification was testimony to a longstanding disease process. After cardiac catheterization the diagnosis entertained was of primary echinococcal (hydatid) cysts involving 2 of the 3 coronary arteries. The other diagnosis was of non-atheromatous coronary artery aneurysms secondary to some kind of long ago coronary

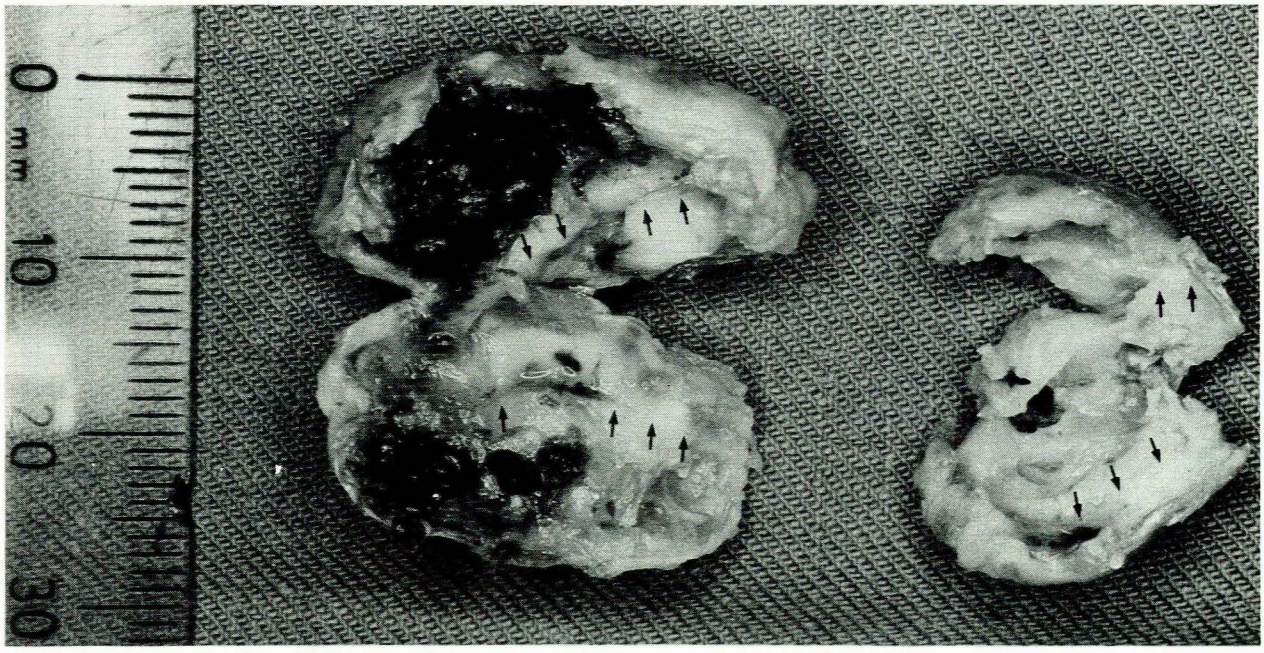

Fig. 6. Excised and bisected calcified masses. The larger one involving the LAD (left) contains a large, friable old thrombus. The smaller mass excised from the RCA (right) also has a small old thrombus. Remnants of the parent coronary artery can be seen to traverse each mass (arrowed).

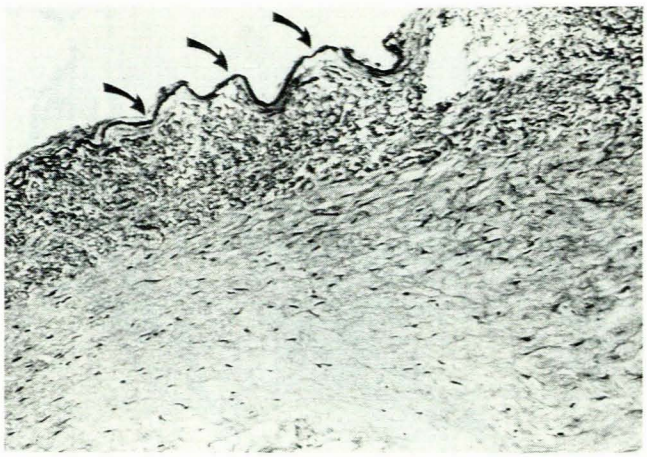

Fig. 8. High-power view of remnants of vessel wall, largely fibrotic. Arrows indicate elastic tissue. Note the absence of atherosclerosis (Verhoeff x 200).

arteritis. At the time of cardiac surgery it was impossible to decide which of these two diagnoses was most likely to be correct.

Coronary arteritis could have been an expression of a systemic disease, in keeping with the patient's previous medical history, but there were no signs of 'activity' of such a disease. Systemic lupus erythematosus is a well-known cause of coronary arteritis[1-3] and can even cause acute MI.[4,5] Other disease states that cause coronary arteritis, with or without aneurysm formation, are rheumatoid arthritis,[6-10] which sometimes results in acute MI,[11] polyarteritis nodosa,[12] mucocutaneous lymph node syndrome or Kawasaki's disease,[13,14] tuberculosis,[15,16] rheumatic fever,[17-21] Takayasu's disease,[22] syphilis[23] (which classically causes an aortitis with coronary ostial stenosis[24]), and viral infection which may either cause acute MI[25] or death.[26-28]

Were the 'masses' coronary artery aneurysms? A point in favour of this is the fact that a definite aneurysm of the first diagonal branch of the LAD coronary artery was delineated. These aneurysms are exceptionally rare,[29-34] the commonest cause being atherosclerosis.[35-38] Congenital coronary artery aneurysms also occur and are often symptomatic.[39,40] Most unusual causes of coronary artery aneurysms are scleroderma,[41] infection,[42] coronary arteriovenous fistula,[43] and other uncertain disease states.[44-47] Excluding arteriosclerotic and congenital coronary artery aneurysms, the great majority are thought to develop after coronary arteritis. The fact that thrombus was found within the masses would be strongly in favour of aneurysms.

Primary echinococcal (hydatid) cysts must be considered in the differential diagnosis. Obstruction to the right ventricular outflow tract by a cyst typically found in the interventricular septum[48] is not uncommon, but involvement of the coronary arteries has only been described once.[49] *Echinococcus granulosus,* the dog tapeworm, usually causes human infection during childhood. The majority of oncospheres (eggs) pass either to the liver by way of the portal system or into the lungs. It is believed that these oncospheres rarely reach the heart via the coronary arteries, and that they never traverse the endocardium from the systemic circulation. Symptoms usually appear by the 3rd or 4th decade and can be caused by allergic response to the hydatid fluid, embolic phenomena, or actual pressure by the cysts. Our patient's symptoms could well have been due to an allergic reaction. She had been eating raw *boerewors* during the previous 5 years, but infestation at that time was not likely to be responsible for the heavily calcified masses. A negative echinococcal complement fixation test does not exclude this

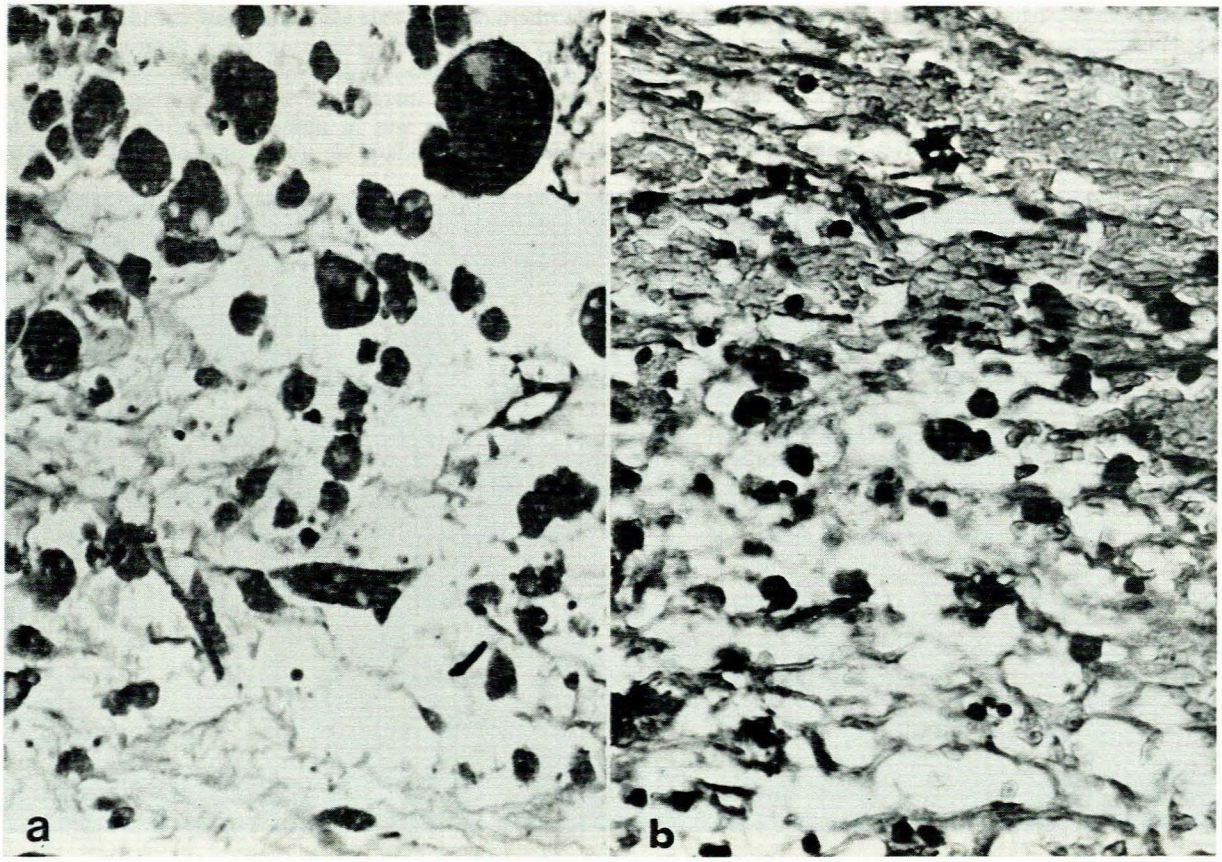

Fig. 9. a — haemosiderin-containing macrophages (H and E x 400); b — scattered lymphoplasmacellular infiltrate (H and E x 400).

diagnosis. Kurban *et al.*[49] of Beirut described a 55-year-old white woman with AP who died in hospital. Autopsy revealed the presence of a 4 cm diameter calcified echinococcal cyst 'overlying the superior part of the interventricular septum on the anterior surface of the heart'. Furthermore, the 'anterior descending branch of the left coronary artery coursed beneath the cyst'. Kurban *et al.*[49] strongly believed that the cyst caused the patient's AP and acute MI. They stated that 'the cyst was lined by a chitinous membrane and contained clear fluid in which scolices and hooklets were found'. A fresh thrombus was noted, as well as evidence of an acute anterior MI and pulmonary embolism. An incidental finding was that of minimal coronary atherosclerosis.

In our patient, remnants of an echinococcus could not be detected within either of the 'cysts' or coronary artery masses, but it is possible for fibrosis and calcification to be the only remnants in this involvement. Haemorrhage into an echinococcal cyst could well give rise to a thrombus. There were numerous plasma cells within the two masses, a feature in keeping with a chronic inflammatory process such as coronary arteritis, or a reaction to a coronary artery aneurysm. This inflammatory response could have been triggered by a low-grade or chronic 'coronary arteritis' secondary to echinococcal infestation.

Kawasaki's disease[13] has been well documented in the adult.[14] Our patient's previous history is certainly not entirely against this diagnostic possibility.

If coronary artery aneurysms, excluding atherosclerotic ones, are left untreated they are said to be possibly dangerous. The operation most commonly carried out is coronary bypass,[50-52] which has even been successful in congenital coronary artery aneurysms complicated by acute MI.[53,54] The large size of the masses in our patient necessitated actual excision together with the parent coronary artery, with closure of both the proximal and distal ends of the transected coronary arteries and re-establishment of blood supply by saphenous vein bypass. The success of the operation in our patient was dramatic. Unfortunately, the precise aetiology of these coronary artery 'masses' will never be determined, but we favour a nonspecific coronary arteritis, particularly Kawasaki's disease, with the later development of coronary artery aneurysms and calcification. Nevertheless, the patient declares that she will no longer devour raw *boerewors*!

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