Anterior myocardial infarction with coronary thrombus formation secondary to acute coronary vasospasm

A case report

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Summary

An acute myocardial infarction in a young man when there was no fixed atherosclerotic lesion in the coronary arteries is reported. Soon after the myocardial infarction the patient was shown to have thrombosis in the anterior descending branch of the left coronary artery but this later disappeared. In view of recurrent angina pectoris, provocation tests were undertaken with ergometrine maleate, cold pressor and hyperventilation. Both the drug and the hyperventilation provoked coronary spasm accompanied by angina pectoris but no ECG evidence of ischaemia.

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Case report

A 22-year-old white serviceman had had an episode of pain resembling angina pectoris at the age of 14 years but no further cardiac symptoms until he was 21 years old in spite of being very active and indulging in competitive sport. He was a heavy smoker.

On 1 May 1984 soon after the end of a rugby match and after-game cold shower he experienced a severe crushing retrosternal pain radiating down his left arm; this was associated with sweating, nausea and dyspnoea. In spite of morphine the pain persisted for 12 hours and was aggravated by deep inspiration and by lying down. The patient's father had died of acute myocardial infarction at the age of 42 years.

On admission to the sick bay the patient was very distressed with tachycardia and frequent ventricular extrasystoles. An ECG (Fig. 1) showed QS waves with ST-segment elevation in V1 and V2, upward-coved ST segments with T-wave inversion in leads V3 and V4, and flat T waves in leads V5 and V6. The diagnosis of an acute anteroseptal myocardial infarction was made; this was supported by serial serum enzyme studies.

On 2 May the patient was transferred to an intensive coronary care unit at 1 Military Hospital, Pretoria. Subsequent ECGs and serum enzyme estimates confirmed the diagnosis of an acute transmural anteroseptal and non-transmural anterolateral myocardial infarction. There were no complications and serum lipid and uric acid levels were normal.

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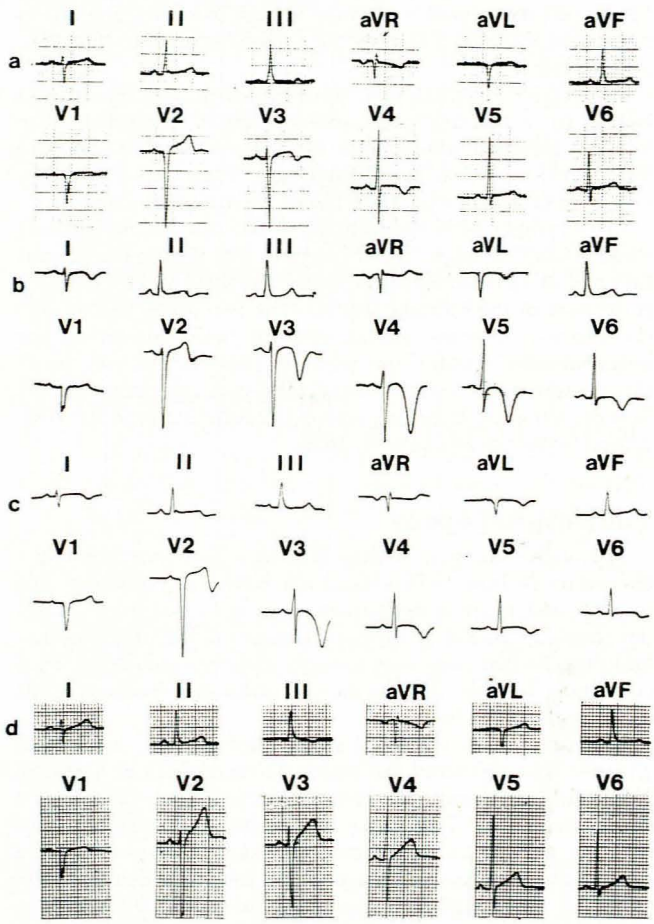

Fig. 1. Resting 12-lead ECG on full standardization: (a) on the first admission showing features of an acute transmural anteroseptal myocardial infarction and possible left posterior hemiblock; (b) 24 hours after admission demonstrating progression to a non-transmural anterolateral myocardial infarction; (c) on the 3rd post-infarction day — an evolving transmural anteroseptal and non-transmural anterolateral myocardial infarction is clearly seen; (d) 16 months after initial infarction — anterior early ventricular repolarization and possible left posterior hemiblock; no definite features of the previous myocardial infarction.

Cardiac catheterization on 9 May 1984 revealed antero-apical segmental dyskinesia and coronary arteriography delineated a normal right coronary artery and a large filling defect within the left anterior descending (LAD) branch of the left coronary artery which was interpreted as an intracoronary thrombus (Fig. 2).

The patient was again catheterized on 20 June 1984 when he had no abnormal symptoms or signs, and on this occasion cine angiography showed a persistently abnormal antero-apical contraction (Fig. 3) but with no signs of intracoronary thrombus (Fig. 4), although there was insignificant narrowing distal to the original site of the thrombus.

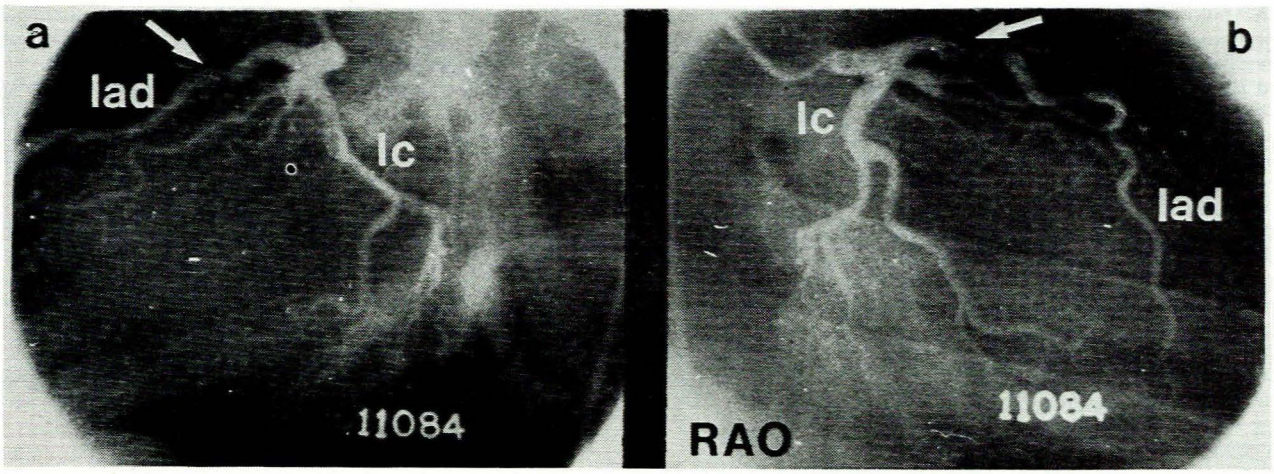

Fig. 2. Left coronary cine angiograms in (a) left anterior oblique (LAO) and (b) right anterior oblique (RAO) views. An intracoronary thrombus is seen (arrowed) in the proximal part of the left anterior descending coronary artery (lad). The left circumflex coronary artery (lc) is free of disease.

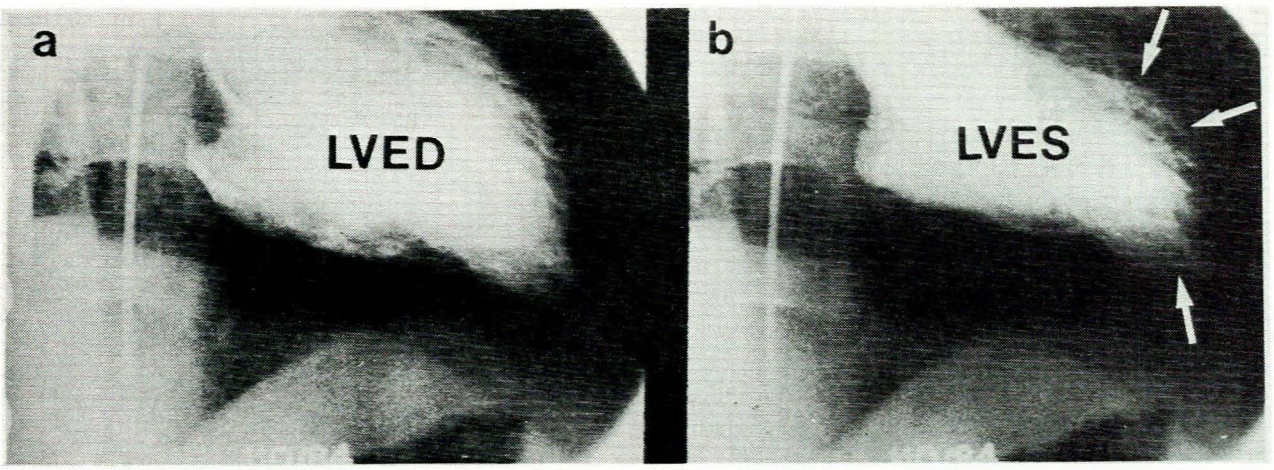

Fig. 3. Left ventricular cine angiograms in the RAO projection showing persistent antero-apical dyskinesia (arrowed): (a) LVED = left ventricle at end-diastole; (b) LVES = left ventricle at end-systole.

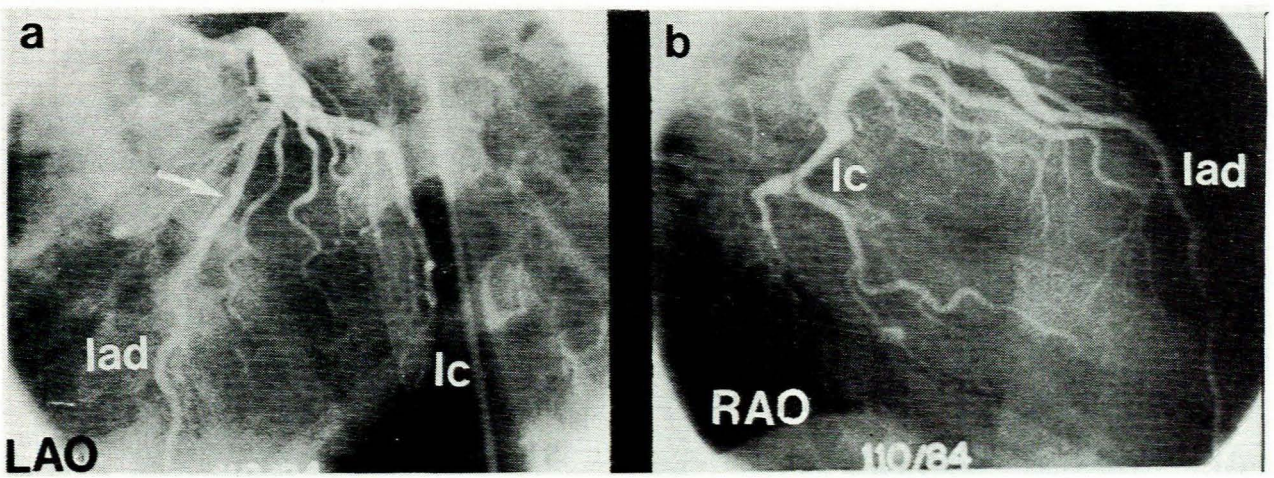

Fig. 4. Left coronary cine angiograms in (a) LAO and (b) RAO projections. The intracoronary thrombus previously seen in the left anterior descending coronary artery is no longer present but there is an insignificant narrowing (arrowed) distal to this site. The left circumflex coronary artery appears normal.

At follow-up he remained well and his drug therapy was discontinued until 10 February 1985, 9 months after the myocardial infarction, when he presented at Tygerberg Hospital having had two 20-minute episodes of retrosternal pain within 24 hours, one during exercise and the other at rest. His ECG demonstrated possible left posterior hemiblock with poor R-wave progression over the anteroseptal leads. Other investigations were negative and he was discharged without treatment.

On 22 August 1985 the patient returned to Tygerberg Hospital with severe precordial pain at rest that had persisted for 3 hours. The ECG now showed somewhat high take-off ST segments in all the anterior leads, possible left posterior hemiblock but nothing else. The results of all other investigations were negative, but in view of the possibility of coronary artery spasm it was decided to perform coronary angiography coupled with ergometrine provocation. The cine angiogram of the left ventricle showed the same abnormality as before but the left coronary artery now had a 30% long-segment stenosis in the LAD branch distal to the site of the previous thrombus (Fig. 5). Repeated injection of ergometrine provoked moderately severe chest pain but no ECG signs of myocardial ischaemia. However, there was evidence of superimposed vasospasm of the 30% LAD branch stenosis giving rise to an 80% reduction in the lumen (Fig. 6). Isosorbide dinitrate sublingually rapidly relieved the pain and further injection of contrast showed disappearance of the vasospasm (Fig. 7). The spasm failed to appear on a cold pressor test but was again provoked on hyperventilation. Isosorbide dinitrate again rapidly relieved the angina and a further cine angiogram demonstrated resolution of the vasospasm.

In view of the demonstration of vasospasm, the patient was given diltiazem 60 mg 3 times daily and advised to use sublingual nitrate when required. A 24-hour Holter study failed to show any ST-segment abnormality or arrhythmia. At his last hospital visit on 23 October 1985 the patient had no symptoms or further signs.

Discussion

The mechanism of this myocardial infarction in a very young man is interesting, since the repeated angiograms show that no fixed significant atherosclerotic lesions existed; hence a functional stenosis such as that caused by coronary artery spasm must be invoked. The role of a cold stimulus in initiating coronary artery spasm must be considered in view of the cold shower before the first episode, but this is negated by the failure of a cold pressor provocation test to precipitate spasm.

The finding of intracoronary thrombus 8 days after the acute myocardial infarction is not surprising since DeWood *et al.*[1] demonstrated the presence of thrombus in 85% of patients

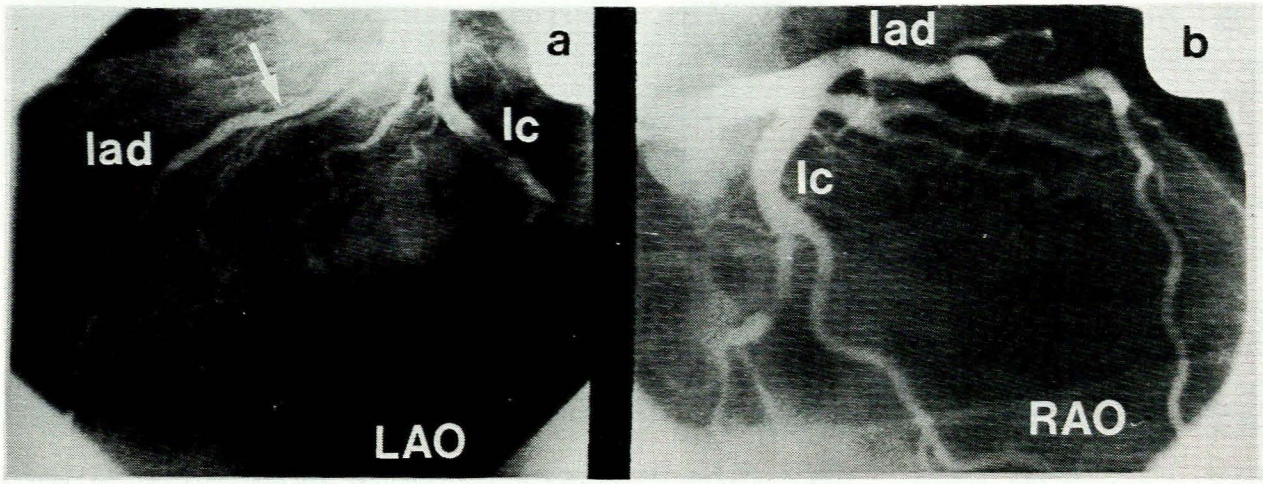

Fig. 5. Left coronary cine angiograms in (a) LAO and (b) RAO views before ergometrine maleate provocation. A long-segment 30% stenosis (arrowed) is seen in the left anterior descending coronary artery distal to the side of the previous intracoronary thrombus. The left circumflex coronary artery is normal.

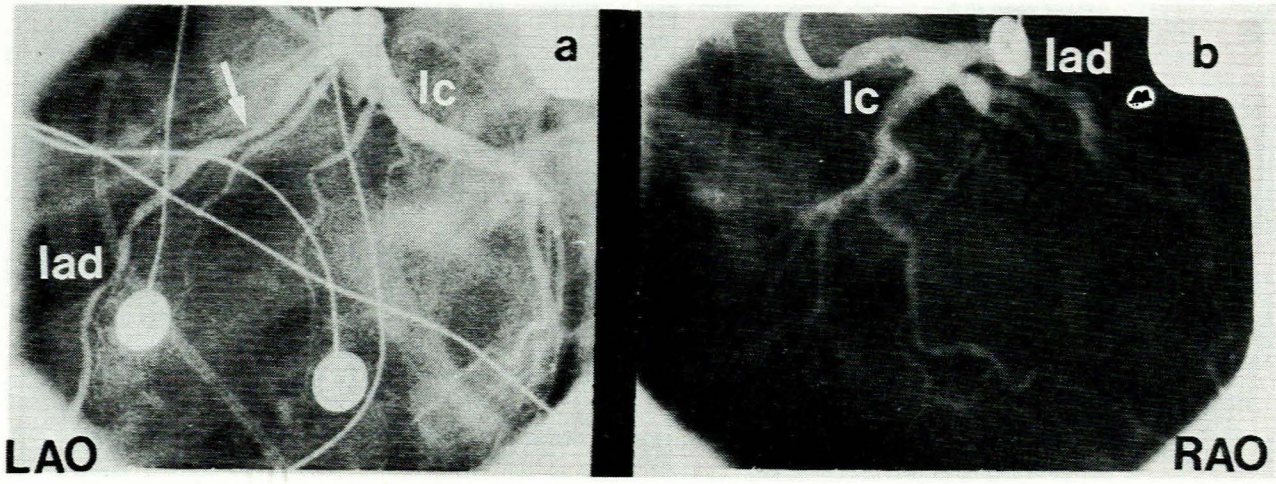

Fig. 6. Left coronary cine angiograms in (a) LAO and (b) RAO projections following a total dose of ergometrine maleate 0,4 mg and during praecordial pain. An 80% stenosis, owing to vasospasm (arrowed), is seen in the left anterior descending coronary artery at the site of the previous 30% 'fixed' stenosis. The left circumflex coronary artery is normal.

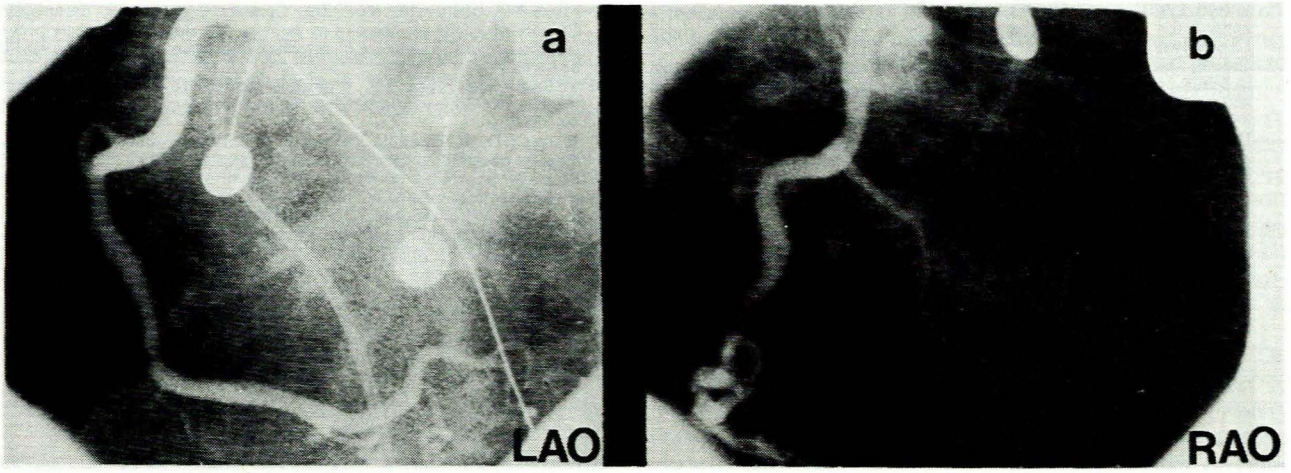

Fig. 7. Right coronary cine angiograms in (a) LAO and (b) RAO views following sublingual nitrate. The vessel is slightly dilated but is free from any focal stenosis.

undergoing coronary arteriography within a few hours of acute myocardial infarction. Of interest in this respect is the recent documentation of coronary thrombi in patients with unstable angina pectoris.[2-5]

While there is no doubt that coronary artery spasm with or without underlying atherosclerotic obstructive coronary artery disease can precipitate acute myocardial infarction,[6-12] the precise mechanism of spasm remains elusive. The fact that the patient experienced angina pectoris again after the acute myocardial infarction is of significance since coronary artery spasm has been incriminated in post-infarction angina.[13]

A number of tests have been introduced to provoke coronary vasospasm during angiography, of which the most specific and sensitive is the ergometrine (ergonovine) maleate injection.[14-17] In our patient this provocation actually precipitated coronary vasospasm while the cold pressor test,[18,19] a controversial method of provoking spasm which was used in this case because of the history of the cold shower, failed to elicit a positive response. This test should probably not be applied as a diagnostic measure in patients with suspected variant angina pectoris.[20] The last provocative test used was that of hyperventilation,[20,21] which increases coronary resistance as does the cold pressor test, and which is known to precipitate vasospasm in susceptible patients. It is also known that hyperventilation is more likely to provoke spasm than a cold pressor test,[22] a finding verified in our patient. To compound the difficulty in understanding the disease mechanisms in our patient, there remains the possibility of synergistic effects of ergometrine maleate, cold pressor and hyperventilation in this particular case.

The demonstration of coronary artery spasm in relation to symptoms of acute myocardial infarction is most important in deciding on appropriate treatment. Beta-blocking drugs can be detrimental to patients suffering from Prinzmetal's angina and the most successful drugs for this type of angina pectoris are the nitrates and calcium antagonists.[23-27] However, management and follow-up of these patients with or without a past history of acute myocardial infarction is difficult because of spontaneous remission,[28,29] for which the mechanism is unknown. Prognosis is greatly dependent on residual myocardial function after the infarct as well as the degree of underlying atherosclerosis. Our patient should have a favourable prognosis in view of the size of the acute myocardial infarction and the virtual absence of atherosclerosis. Nevertheless coronary vasospasm can give rise to recurrent acute myocardial infarction[30] and our patient will be followed up and continued on a calcium antagonist for some time.

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Blastomycosis

A case report

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Summary

Blastomycosis was once thought to be restricted to North America; it has rarely been found in the RSA. The first case reported in the RSA was associated with a diffuse pulmonary infiltrate and cavitation; the patient responded to amphotericin B.

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Blastomycosis is an infection caused by the fungus *Blastomyces dermatidis*. The disease was once thought to be restricted to North America. While cases have been reported in Africa[1,2] and the northern parts of South America,[2] it remains an uncommon illness.

We believe this is the first reported case in the RSA of a diffuse pulmonary infiltrate associated with blastomycosis.

Case report

A 47-year-old diamond prospector was admitted to an outlying hospital with a diagnosis of suspected tuberculosis because of the presence on radiography of pulmonary infiltrate. A number of sputum specimens were negative for tuberculosis and he was transferred to Johannesburg Hospital for biopsy of an enlarged right cervical lymph node.

There was a 6-month history of fever, malaise, weight loss and a cough productive of purulent sputum. The patient had become progressively more dyspnoeic over this period. Examination revealed him to be wasted, tachypnoeic and cyanosed. Bilateral tonsillar and right cervical adenopathy were present. Diffuse bilateral crackles were audible on examination of the chest. A chest radiograph showed the presence of a bilateral nodular pulmonary infiltrate with evidence of cavitation (Fig. 1).

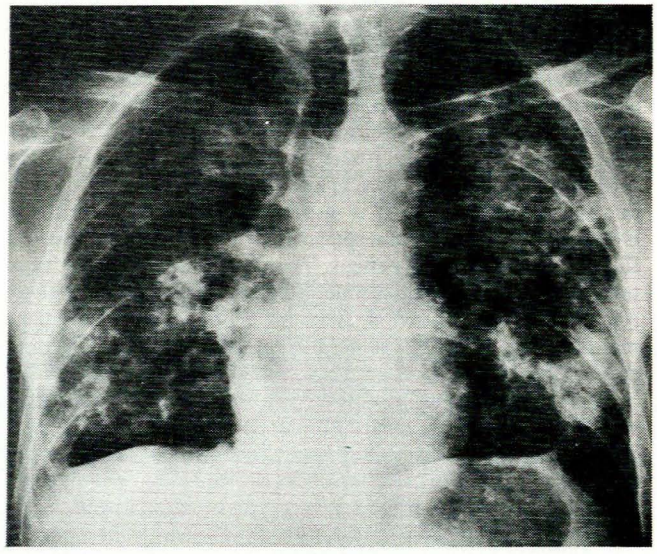

Fig. 1. Chest radiograph on admission.

The haemoglobin value was 14,1 g/dl and the white cell count 17,0 x 10^9/l. The erythrocyte sedimentation rate was 113 mm/1st h (Westergren). Sputum culture for *Mycobacterium tuberculosis,* other bacteria and fungi was repeatedly negative. Histological examination of a right cervical lymph node showed reactive changes only, and of a biopsy specimen from the right tonsil showed epithelioid granulomas, micro-abscesses and multiple spherical fungal organisms varying in diameter, and with doubly refractile capsules,

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