

# Acute myocardial infarction due to coronary vasospasm secondary to industrial nitroglycerin withdrawal

## A case report

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### Summary

A case of acute transmural anterior myocardial infarction in a 45-year-old Black employee of an explosives factory during a period of withdrawal from industrial nitroglycerin is documented. Angiography revealed that the patient had normal coronary arteries. Coronary vasospasm could not be induced by the ergometrine (ergonovine) maleate provocation test. It is postulated that the infarction was directly attributable to coronary vasospasm provoked by the 'industrial nitroglycerin withdrawal syndrome', since there was no evidence of any other non-atheromatous aetiological factor. The authors believe this to be the first such case in a Black subject reported in the literature.

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### Case presentation

The patient was a 45-year-old Black man employed in an explosives factory where he had been handling industrial nitroglycerin for some years. A routine resting ECG in May 1981 revealed sinus rhythm and features of early ventricular repolarization compatible with the 'normal variant pattern' (Grusin type II) encountered in this racial group

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(Fig. 1a). A nonspecific intraventricular conduction defect was also noted. The patient's only risk factor for ischaemic heart disease was that he smoked some 10 cigarettes daily. He was completely asymptomatic until about 07h00 on Sunday 22 August 1982, i.e. some 36 hours after the last exposure to industrial nitroglycerin, when he experienced a sudden severe crushing retrosternal pain while he was busy digging in the garden. This pain radiated to the right shoulder and down the forearm and was associated with profuse sweating and dyspnoea. At this stage he did not seek medical aid and continued to experience the chest pain for about 13 hours, when it slowly decreased in severity. The following morning (23 August) he approached the doctor in the factory hospital, who found him normotensive with no evidence of cardiac decompensation. A resting ECG (Fig. 1b) revealed the features of an acute transmural anteroseptal and anterolateral myocardial infarction. Serum enzyme levels were elevated (Table I). The patient was

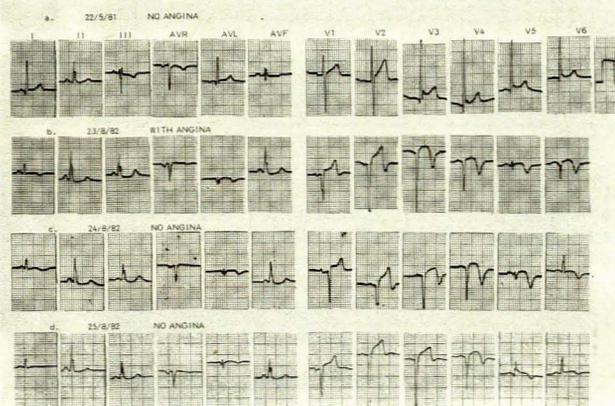


Fig. 1. Resting ECG (full standardization): a — PR interval 0,15 second; mean QRS axis  $-5^{\circ}$ , early ventricular repolarization in anterior leads in keeping with the 'normal variant pattern' (Grusin type II); b — features of acute transmural anteroseptal and non-transmural high lateral myocardial infarction (taken during angina); c — evolving myocardial infarction recorded in absence of angina; d — further evolution of the myocardial infarction.

TABLE I. SERUM ENZYME VALUES

Enzyme*	23 Aug.	24 Aug.	24 Aug.	25 Aug.	26 Aug.	30 Aug.	Normal range
CK	1 290	275	177	99	74	20	0 - 50
CK-MB	161	6	—	—	—	—	< 10
AST	221	85	161	105	76	41	0 - 40
ALT	—	—	98	116	87	27	0 - 53
LDH	1 728	1 043	1 090	1 003	762	446	100 - 350

\*All values in U/l.

CK = creatine kinase; CK-MB = MB iso-enzyme fraction of CK; AST = aspartate transaminase; ALT = alanine transaminase; LDH = lactate dehydrogenase.

admitted to the factory hospital and started on oral nitrate therapy and heparin. On the following morning he was transferred to the Intensive Coronary Care Unit at Tygerberg Hospital. His blood pressure was 120/80 mmHg, and his pulse was regular and normal (84/min); a loud fourth heart sound was noted, but there were no features of cardiac failure. A chest radiograph was normal and a resting ECG confirmed the diagnosis of an acute transmural anteroseptal and non-transmural anterolateral myocardial infarction (Fig. 1c). The patient was monitored but no arrhythmias were detected. Administration of high doses of oral nitrates as well as intravenous heparin was continued. The patient had no further episodes of chest pain, and serial ECGs (Figs 1d and 2a-c) and serum enzyme values (Table I) over the succeeding days demonstrated the classic features of an evolving myocardial infarction. Results of other routine biochemical and haematological investigations were within the normal range. Tests for possible underlying syphilis (VDRL and rapid plasma reagin tests) were negative. Some 5 days after the initial episode of chest pain a <sup>99m</sup>Tc-pyrophosphate ('hot spot') scan demonstrated increased uptake in the anterolateral and apical regions, in keeping with a recent myocardial infarction.

On 30 August, 1 week after his acute myocardial infarction,

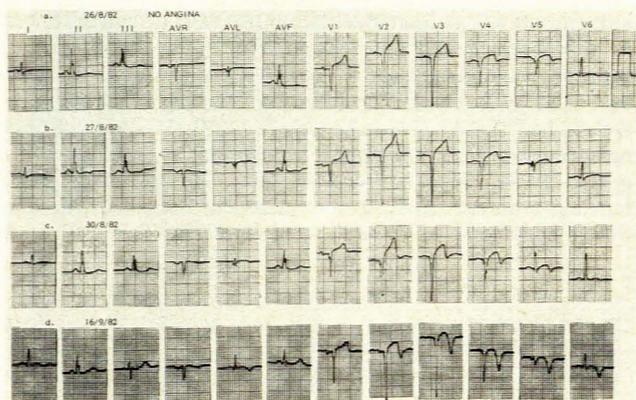


Fig. 2. ECG during angina-free period: a-c — showing evolution of transmural anteroseptal myocardial infarction with non-transmural anterolateral component; d — ECG 2 weeks after the acute myocardial infarction, demonstrating the completed infarction.

cardiac catheterization and selective coronary arteriography were undertaken by the Seldinger technique via the groin. All the intracardiac pressures and indices of left ventricular function were normal but left ventricular cine angiography in the right anterior oblique projection displayed akinesia of the anterolateral and apical regions secondary to infarction (Fig. 3). Baseline selective coronary arteriography in multiple views delineated a normal dominant right coronary artery (Fig. 4) and left coronary artery (Fig. 5). The patient did not complain of any chest pain during the coronary artery injections and no electrocardiographic changes were noted in standard lead II and lead V5 on the oscilloscope. A 12-lead ECG at this stage was no different from the previous tracings. The ergonovine (ergometrine) maleate provocation test was carried out in an attempt to document coronary vasospasm as a possible factor causing his myocardial infarction. This was done by the injection of an initial bolus of 0,025 mg into the main pulmonary artery while monitoring the aortic pressure and standard lead II and lead V5 on the oscilloscope. In addition, a 12-lead ECG was recorded every minute. A further bolus of 0,025 mg was given after 4 minutes and the monitoring procedure was repeated; boluses of 0,05 mg were then administered to a total of 0,4 mg ergonovine but no changes were noted at any stage during this test. Repeat selective coronary arteriography in multiple projections failed to show any possible provoked spasm of either the right or the left coronary artery, and the patient did not complain of any angina. The procedure was completed without complication. Serial serum enzyme estimations and 12-lead ECGs following cardiac catheterization showed no additional changes.

After a few days the patient was transferred back to the factory hospital to convalesce. He was treated with isosorbide dinitrate 40 mg 8-hourly and nifedipine 10 mg 8-hourly orally. Repeated serial ECGs demonstrated evolution of the transmural anteroseptal and non-transmural anterolateral myocardial infarction (Fig. 2d).

A diagnosis of vasospasm involving the left coronary artery secondary to industrial nitroglycerin withdrawal, severe enough to culminate in acute myocardial infarction, was therefore established. No underlying atherosclerotic plaques could be demonstrated angiographically. The patient was withdrawn from exposure to nitroglycerin and continues to be free of angina pectoris on his present medication.

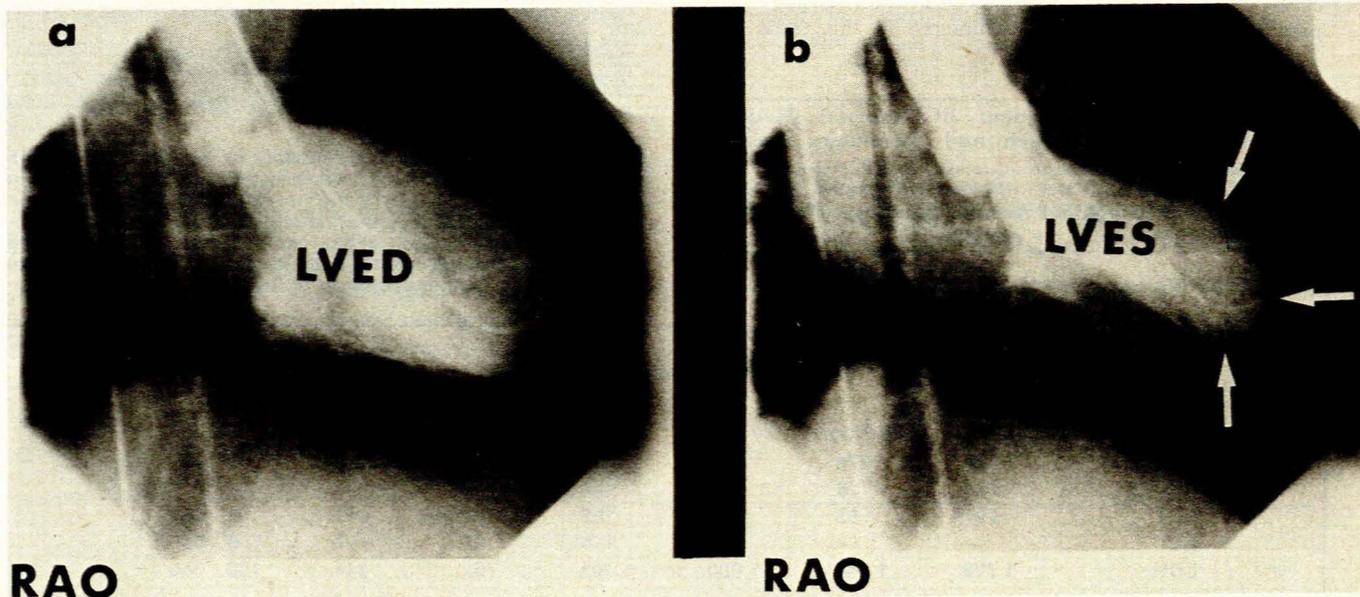


Fig. 3. Left ventricular cine angiograms in the right anterior oblique projection illustrating anterolateral and apical akinesia secondary to myocardial infarction (arrowed): a — left ventricle in end-diastole; b — left ventricle in end-systole.

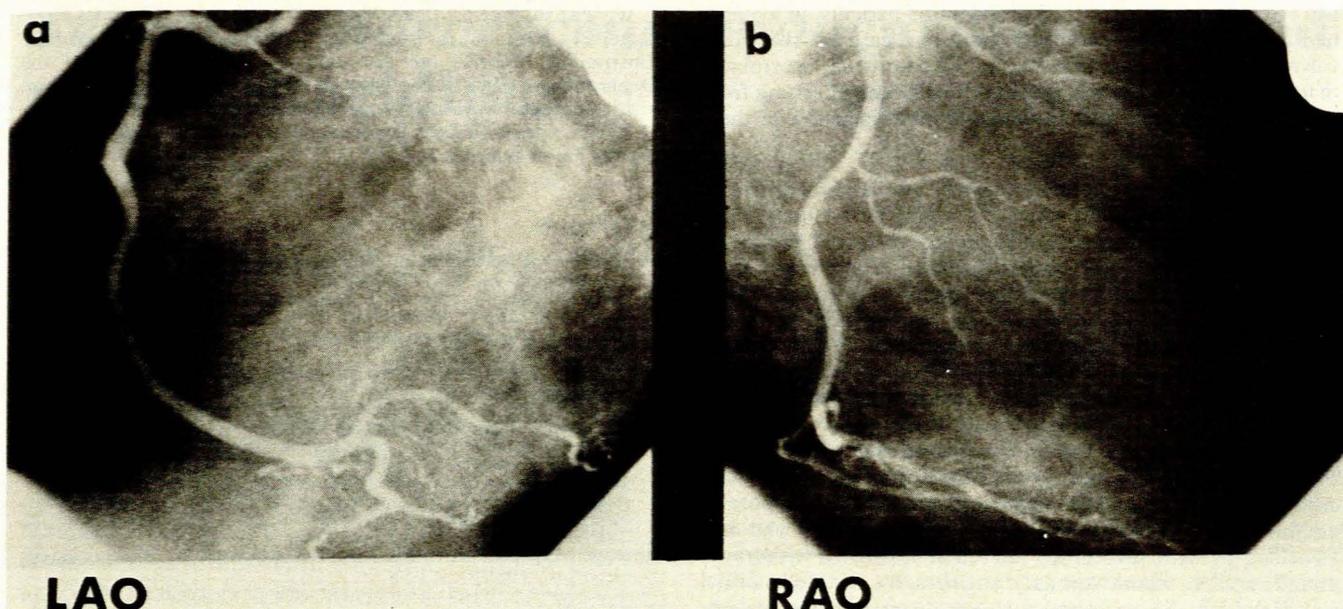


Fig. 4. Right coronary artery cine angiograms in (a) left anterior oblique and (b) right anterior oblique projections. The vessel is dominant and free of any obstructive lesions.

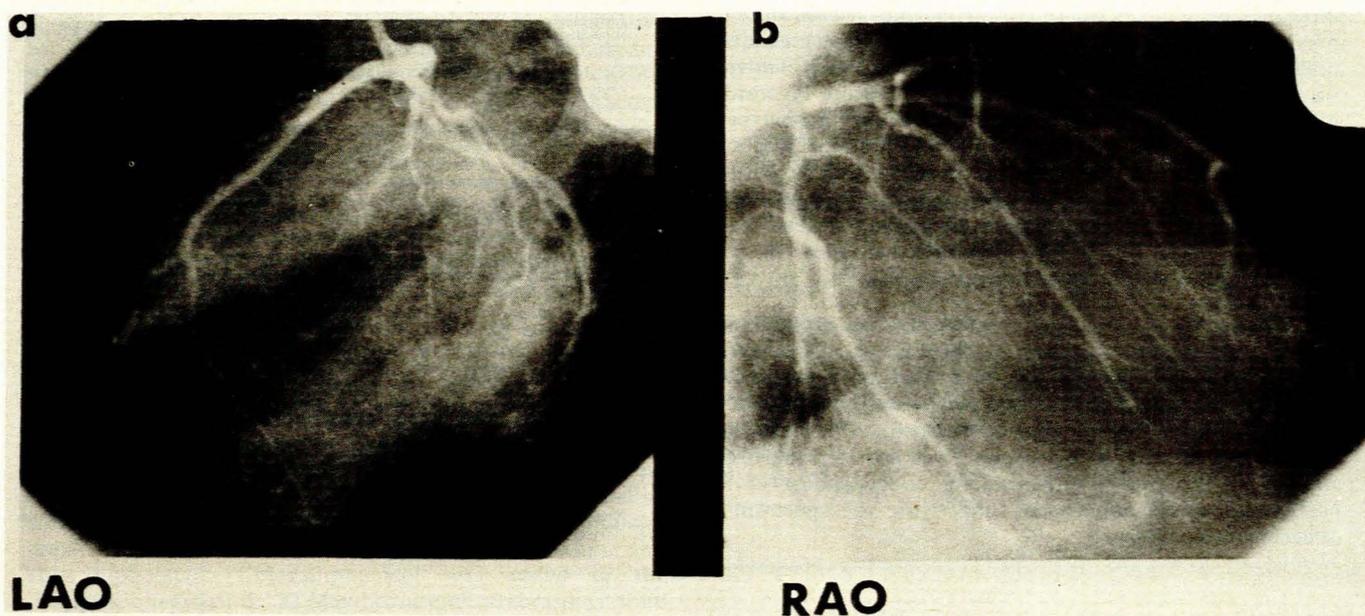


Fig. 5. Left coronary artery cine angiograms in (a) left anterior oblique and (b) right anterior oblique projections. The left mainstem, left anterior descending and left circumflex vessels appear normal.

## Discussion

Atheromatous coronary artery disease has been considered a rarity in the Black population of South Africa.<sup>1-3</sup> Seftel<sup>4</sup> was not surprised by this fact as hypertension and obesity, both common in the Black, are not considered to be significantly atherogenic. It is generally accepted that ischaemic heart disease is more common in the urban Black than in his rural counterpart,<sup>5</sup> but Chesler *et al.*<sup>6</sup> did not believe this to be the case. Postmortem evidence of obstructive coronary atherosclerosis and myocardial infarction in Blacks older than 30 years has been rare. Thus Becker<sup>7</sup> found a 1,5% incidence, and both Higginson *et al.*<sup>8</sup> and Kallichurum<sup>9</sup> found a 2,2% incidence. Chesler *et al.*<sup>6</sup> stated that

the disease was probably even less frequent and estimated a prevalence rate of less than 0,05% of medical admissions.

Acute myocardial infarction, one of the consequences of the spectrum of ischaemic heart disease, is therefore uncommon. In an 11-year period (1951-1961) Seftel *et al.*<sup>10</sup> found only 30 cases of myocardial infarction at Baragwanath Hospital. Chesler *et al.*<sup>6</sup> were the first to document the coronary angiographic features in Black subjects with clinical and electrocardiographic characteristics of myocardial infarction. These workers reported on 13 cases in which possible syphilis, viral myocarditis, collagen disease and dissecting aortic aneurysm were excluded; 3 of the patients were found to have single-vessel disease affecting the right coronary artery, 5 had diffuse double-vessel disease and 2

had triple-vessel disease. The remaining 3 patients, of whom 2 had documented prolapse of the posterior leaflet of the mitral valve, had normal coronary arteries. Chesler *et al.*<sup>11</sup> postulated that either coronary artery spasm or fibrin emboli arising from the redundant mitral valve apparatus was responsible in these latter patients.

Non-atheromatous coronary artery disease such as is encountered in the collagenoses,<sup>12,13</sup> syphilitic coronary ostial stenosis<sup>14,15</sup> and aortic arteritis (Takayasu's disease)<sup>16</sup> has always to be considered in the differential diagnosis of angina and acute myocardial infarction in the Black population group. Lange *et al.*<sup>17</sup> gave a classic description of non-atheromatous ischaemic heart disease consequent upon withdrawal from chronic exposure to industrial nitroglycerin and noted the presence of spontaneously occurring coronary vasospasm, as well as acute myocardial infarction. Klock<sup>18</sup> was the next to report on spontaneous coronary artery spasm during angiography. Przybojewski and Heyns<sup>19</sup> documented the first case of this entity occurring in a Black in South Africa. We now report on another Black employee of an explosives factory who had sustained an acute myocardial infarction during the withdrawal period, and in whom coronary angiography revealed normal vessels which did not exhibit spasm on ergonovine maleate provocation. The fact that this investigation was carried out some 10 days after withdrawal from industrial nitroglycerin may explain why vasospasm could not be induced. However, it may be that the pathogenetic mechanism of coronary artery spasm in these patients is not influenced by ergonovine maleate, since the previous patient reported by us<sup>19</sup> had a negative ergonovine provocation test. The present patient also proved to be of interest since his initial ECG (Fig. 1a), before the acute myocardial infarction, displayed features in keeping with the Grusin type II pattern.<sup>20</sup> It must be emphasized that the differential diagnosis should include such conditions as acute pericarditis, chronic constrictive pericarditis, myocardial infarction, myopericarditis, cardiomyopathy, left ventricular aneurysm, athletic heart syndrome and such miscellaneous entities as the hyperventilation syndrome and hyperkalaemia.<sup>19,21-23</sup>

The reason for reporting this case is to illustrate another cause for acute myocardial infarction in Black South Africans. The pathophysiology of the 'industrial nitroglycerin withdrawal syndrome' has been fully discussed previously,<sup>19</sup> and will not be reiterated. This entity presents a therapeutic challenge, and research into the various aspects may well provide some of the answers to the problems bedeviling understanding of the pathophysiological mechanisms underlying the spectrum of ischaemic heart disease.

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