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Acute transmural myocardial infarction — coronary vasospasm, thrombosis or coronary embolus?

A case report

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Summary

A very fit 28-year-old Coloured athlete presented with an acute transmural anteroseptal and non-transmural anterolateral myocardial infarction (MI). He had no significant risk factors for coronary artery disease apart from moderate cigarette smoking. Cardiac catheterization 2 months later demonstrated a significant area of myocardial damage as well as a large mural thrombus, but the coronary arteries appeared normal apart from a large irregular filling defect in the proximal left anterior descending (LAD) branch, apparently due to a thrombus. Cardiac catheterization a further 4 months later documented no further filling defect in the LAD branch and the coronary arteries appeared free of disease. Ergometrine maleate provocation on this occasion failed to demonstrate any coronary vasospasm. Possible pathophysiological mechanisms for the unexpected MI are outlined.

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Case report

The patient was a married 28-year-old Coloured man who smoked less than 10 cigarettes per day and had no family history of ischaemic heart disease. He classed himself as an athlete and claimed to have been exceptionally healthy until 28 December 1982 when he developed a severe crushing retrosternal pain while walking fairly briskly. This symptom lasted some 30 minutes and was associated with nausea and vomiting. He was taken to a district hospital where a resting ECG was interpreted as showing an 'extensive anterior myocardial infarction' (MI). This diagnosis was apparently confirmed by serial serum enzyme estimations and he was treated with heparin and oral isosorbide dinitrate. He suffered no further angina until 9 days after admission when a repeat ECG demonstrated 'complete right bundle-branch block' (RBBB). It was therefore decided to transfer him to our Intensive Coronary Care Unit on 7 January 1983. On admission the only abnormal findings were a loud fourth heart sound at the apex and a widely split second heart sound at the base. A resting 12-lead ECG (Fig. 1) showed sinus rhythm of 80/min, a P-R interval of 0,14 second and a mean QRS axis of -40°. There were also features of bifascicular heart block (left anterior hemiblock and complete RBBB) and of a recent transmural anteroseptal and non-transmural anterolateral MI. A chest radiograph was normal, as were results of laboratory studies.

At this stage it was decided against insertion of a temporary right ventricular cardiac pacemaker. ECG monitoring revealed no arrhythmias; daily resting ECG tracings continued to show

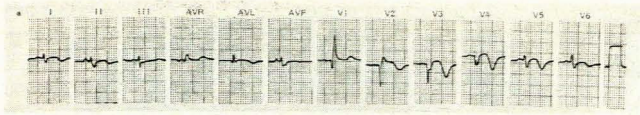

Fig. 1. Resting 12-lead ECG on full standardization — features of a recent transmural anteroseptal and non-transmural anterolateral MI are seen and bifascicular block (left anterior hemiblock and complete RBBB) is also present.

bifascicular heart block but at no stage complete heart block. Serum enzyme measurements were all normal and the patient never complained of angina. Heparin infusion therapy was continued for 5 days and simultaneous warfarin treatment was begun. In view of the patient's youth and the lack of risk factors for atheromatous ischaemic heart disease, the possibility of non-atheromatous coronary artery disease was investigated. Collagen screening, serological testing for syphilis and tests for auto-immune disease were all negative. The patient was progressively mobilized and on 21 January 1983, the day of his discharge, a submaximal effort test (modified Bruce protocol) failed to elicit any ST-T-wave segment change or precipitate angina. However, early in the recovery period numerous unifocal ventricular extrasystoles as well as long runs of ventricular bigeminy were seen. Because of this the patient was given atenolol 100 mg/d in addition to isosorbide dinitrate 40 mg 3 times daily and warfarin. He was discharged after having been in hospital for 2 weeks.

After 4 weeks the patient was assessed at the outpatient department when he complained of several episodes of atypical chest pain. Clinical findings were unchanged, as was the resting ECG. A 24-hour Holter monitor demonstrated very occasional unifocal ventricular extrasystoles. Since the patient had suffered an acute MI at such a young age and wished to recommence jogging, he was admitted for cardiac catheterization on 24 February 1983, approximately 2 months after the MI. This procedure was carried out by the Seldinger technique. All the intracardiac pressures and indices of left ventricular function were normal. However, a left ventricular cine angiogram in the right anterior oblique (RAO) projection delineated extensive antero-apical akinesia secondary to the previous MI as well as a large apical mural thrombus (Fig. 2). The rest of the left ventricle contracted normally and there was no evidence of mitral insufficiency or mitral valve prolapse. Selective coronary angiography in the RAO and left anterior oblique (LAO) views demonstrated a normal dominant right coronary artery (Fig. 3). The left coronary artery gave rise to a normal left circumflex branch. A most striking finding was the presence of a large irregular filling defect in the left anterior descending (LAD) branch just distal to the first septal perforator but proximal to the first diagonal branch, best visualized on the RAO view (Fig. 4b). This defect appeared to have the characteristics of a thrombus, but the rest of the vessel seemed normal. The patient had no angina or arrhythmias during angiography. Soon afterwards he was discharged with no change in his medication.

Regular visits to the Cardiac Clinic found him still complaining of atypical chest pain not always relieved by sublingual isosorbide dinitrate. Repeated resting ECGs revealed no new features and clinical examination always recorded a loud fourth heart sound. Anticoagulation was well controlled. Several Holter-monitoring sessions failed to demonstrate any arrhythmia. A fasting lipogram 5 months after the MI was normal. Repeat submaximal effort testing while the patient was taking nitrates and a ß-blocker failed to show any myocardial ischaemia or provoke arrhythmia or angina. A technetium-99m pyrophosphate gated blood pool scintiscan delineated features of a large antero-apical left ventricular aneurysm with a reduced ejection fraction of 29%. It was decided to readmit the patient on 25 May 1983 (6 months after the MI) in order to recatheterize him with the object of detecting coronary artery spasm. Left ventricular cine angiography in the RAO projection again demonstrated the apical mural thrombus, which appeared to be smaller than before; the contraction abnormality was unchanged. Since bifascicular heart block was present a temporary prophylactic right ventricular pacemaker electrode was inserted before coronary angiography. Right coronary angiography demonstrated a normal dominant vessel. Selective left coronary injection in multiple projections now delineated normal left circumflex and LAD branches; the thrombus in the LAD branch was no longer evident. The ergometrine maleate provocation test was then carried out with an initial bolus of 0,025 mg into the main pulmonary artery while monitoring the aortic pressure and standard leads II and aVL. A further bolus of 0,025 mg was given after 4 minutes followed by boluses of 0,05 mg until a total of 0,40 mg of ergometrine maleate had been given. The patient had no angina, no signs of myocardial ischaemia and no arrhythmias or adverse haemodynamic reaction. Selective coronary angiography demonstrated

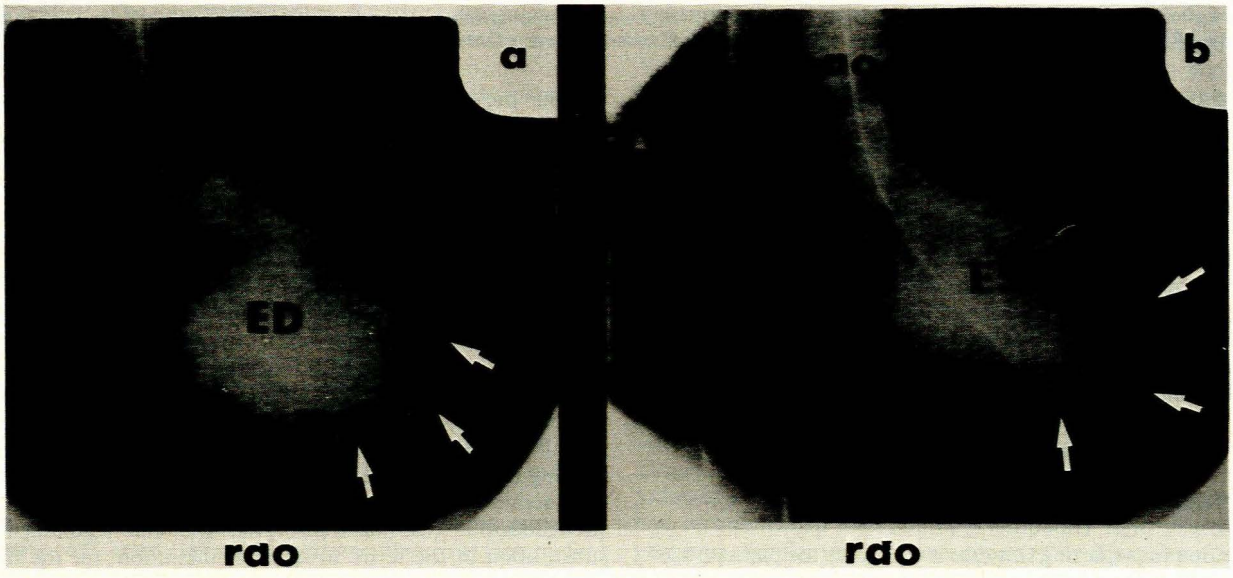

Fig. 2. Left ventricular cine angiograms in the RAO projection delineating antero-apical akinesia and a large mural thrombus (arrowed) **secondary to previous MI (a — ED = end-diastole; b — ES = end-systole; ao = ascending aorta).**

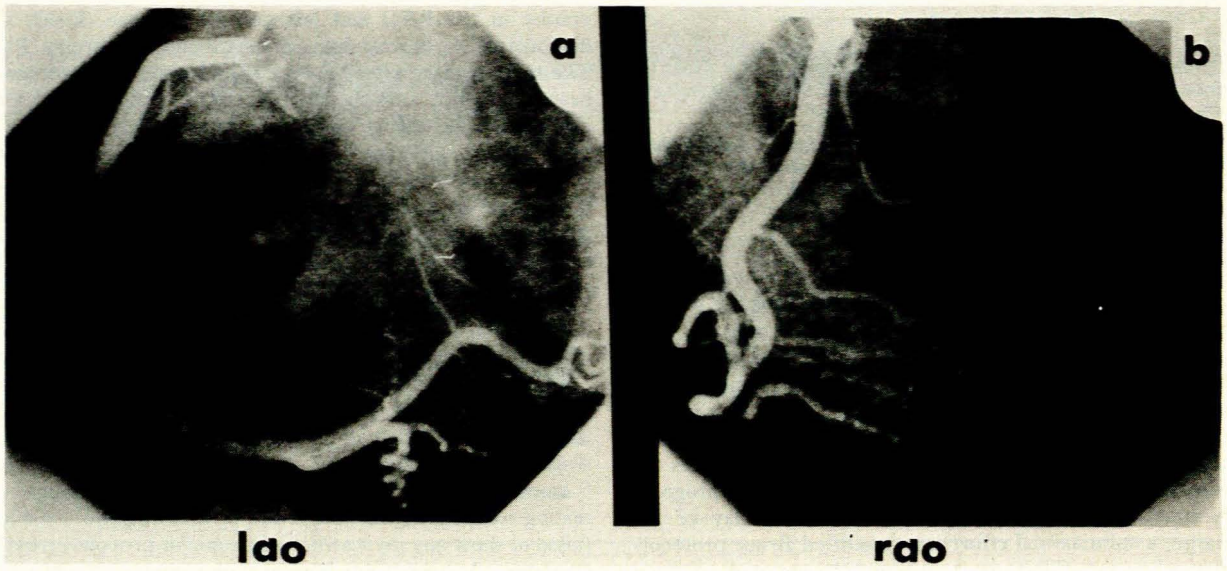

Fig. 3. Right coronary cine angiograms in the (a) LAO and (b) RAO views. The vessel is dominant and appears to be free of disease.

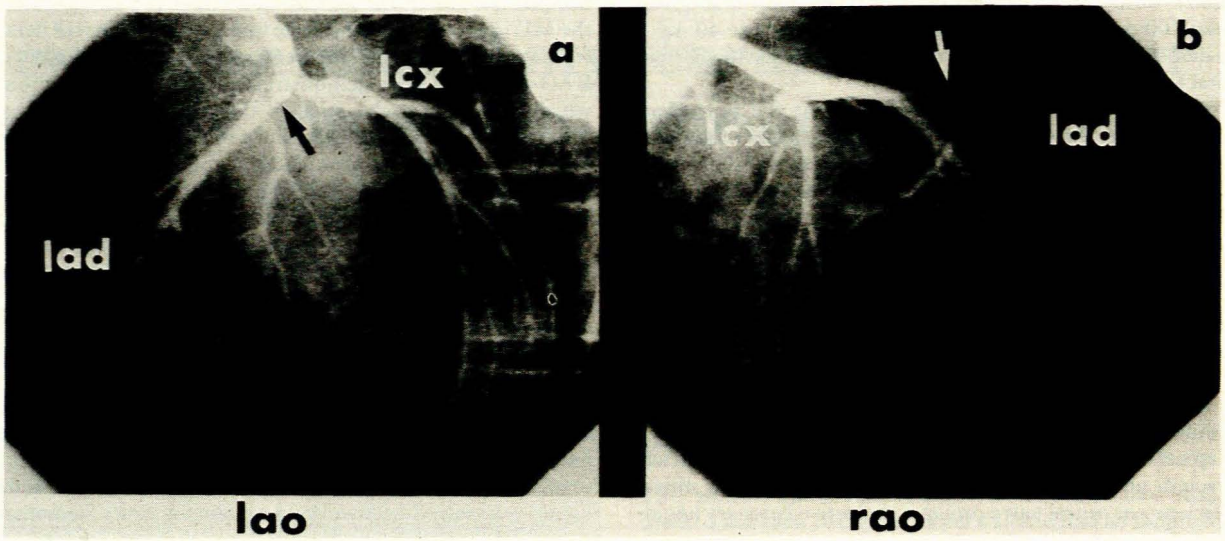

Fig. 4. Left coronary cine angiograms in the (a) LAO and (b) RAO views demonstrating a large irregular filling defect (arrowed) **in the proximal LAD branch. The LAD itself and the left circumflex (lcx) branch show no other visible lesions.**

slight diffuse narrowing of the coronary circulation but failed to reveal any localized vasospasm.

The insertion of a prophylactic permanent cardiac pacemaker was discussed with the patient but postponed at his wish. He was soon discharged, receiving a ß-blocker, nitrate and warfarin. I last saw him on 27 July 1983 when he denied having any symptoms; physical examination revealed a loud fourth heart sound but nothing else abnormal. A resting ECG delineated the previous bifascicular heart block (complete RBBB and left anterior hemiblock), as well as an old transmural anteroseptal and non-transmural anterolateral MI. He had not smoked a cigarette since the MI and was again jogging regularly.

Discussion

The reason for reporting this case is to discuss the pathophysiological sequence of events and to attempt to gain further understanding of the vexed question of what triggers an acute MI. Classic and orthodox thinking has centred around the need for the presence of a significant obstructive atherosclerotic coronary lesion initially. Most pathologists will accept that an underlying atherosclerotic plaque in a coronary artery will, given the correct precipitating factors, be involved in a dissection or rupture with ensuing thrombus formation and coronary occlusion.[1,2] Thus, the concept of a physical obstruction of a coronary artery has been indelibly printed on our minds. As long ago as 1912, when Herrick published his classic paper entitled 'Clinical features of sudden obstruction of the coronary arteries',[3] the concept of a 'coronary thrombosis' became almost synonymous with 'acute MI'. This was unfortunate since other avenues of thought were temporarily inhibited.[4] Most of these patients, however, had documented 'transmural' MIs. Some 25 years later Friedberg and Horn[5] added more fuel to the pathological fire by proving that an MI could ensue without objective proof of an obstructive coronary thrombosis. They showed the presence of subendocardial (or non-transmural) MI and recommended substitution of the term 'myocardial infarction' for the more classic 'coronary thrombosis' since their findings clearly demonstrated that these two were not interchangeable. This suggestion was amplified by their postulating possible 'coronary spasm' in

the pathophysiology.[5] This concept, although not completely new,[6] again made room for more critical and objective thought. Further support for a 'vasospastic mechanism' involving the coronary artery vasculature was strengthened by the absence of thrombi in coronary arteries at autopsy soon after an acute MI.[5]

This raised the critical and important question whether coronary thrombosis was the result rather than the cause of MI and therefore a secondary event. Nevertheless, much further research cast doubt on the exact interrelationship of acute MI and coronary thrombosis, and Chandler *et al.*[7] were obliged to claim that 'a substantial body of knowledge supports the classic concept of the primary role of thrombosis in the pathogenesis of infarction'. This statement is further amplified by the claim of Muller[8] in 1983 that 'the major riddle of the role of coronary thrombosis in infarction has been solved: thrombosis is a prominent causative factor in the vast majority of cases of transmural infarction'. Muller[8] further claimed that 'it is legitimate to revive usage of the diagnosis of "coronary thrombosis" for many patients' and that 'in a patient with a fresh occlusion, this diagnosis may be more accurate than "myocardial infarction" because immediate restoration of flow could lead to salvage of all the ischemic myocardium without infarction'.

Respected workers in this field over approximately the last decade have emphasized the importance of a vasospastic aetiology.[9-11] Thus, the concept of 'fixed stenosis' has been downgraded and the idea of 'functional stenosis' has been accepted more readily. This change was made possible by the more frequent employment of selective coronary angiography in patients with unstable angina pectoris ('pre-infarction angina') before or after MI. The role of coronary artery spasm in the aetiology of unstable angina pectoris is now considered to be significant, and even more so in Prinzmetal's variant angina.[12] The interrelationship between coronary artery spasm and coronary artery thrombosis remains controversial. Most recently, Zelinger *et al.*[13] reported a case of variant angina culminating in acute MI in which cardiac catheterization demonstrated a coronary thrombosis some 6 hours after the estimated commencement of the MI. I recently reviewed the role of Prinzmetal's angina in the causation of acute MI.[14] That coronary vasospasm can give rise to coronary thrombosis with subsequent acute MI appears to have been shown conclusively by Vincent *et al.*[15] in a recent publication. These authors state that 'this spasm-thrombosis sequence may explain the occurrence of myocardial infarction in occasional, often young, patients, such as athletes who have no recognizable coronary-artery disease on subsequent examination'. The enigmatic finding of 'myocardial infarction with angiographically normal coronary arteries' is well known to coronary arteriographers.[16]

During the past few years more active and aggressive interventional techniques have further contributed to our understanding of the mechanisms involved in acute MI. More specifically, selective coronary arteriography within hours of the onset of an acute transmural MI has demonstrated the presence of fresh coronary artery thrombus in approximately 85% of patients in whom thrombolysis by intracoronary streptokinase infusion was contemplated.[17] On the other hand, the coexistence of coronary vasospasm, as demonstrated by reperfusion after intracoronary nitroglycerin infusion, has varied between 2%[18] and 40%.[9] Introduction of percutaneous transluminal coronary angioplasty in patients with unstable angina pectoris and, more recently, application of this technique in conjunction with intracoronary thrombolysis by streptokinase in acute transmural MI, has further highlighted the occurrence of vasospasm which may have preceded thrombosis.

An attempt to elucidate the cause of my young patient's extensive MI must take into account the risk factors for atherosclerotic coronary artery disease. The fact that he was a Coloured man with no family history of ischaemic heart disease, had a normal lipid profile and was athletic weighed heavily against the likelihood of underlying atherosclerosis, but cigarette smoking may have played an important role. Coronary atherosclerosis in Coloured patients has been previously described in association with cigarette smoking and features of 'coronary intimal fibrous stenosis'.[19] Dietary factors in this population group were thought to modify the pathological findings. Non-atheromatous causes of acute MI in the young Coloured male, such as polyarteritis nodosa[20] and syphilitic coronary ostial stenosis,[21] must always be considered in the differential diagnosis. Of further interest is the documentation of multiple coronary thrombosis in 'normal' coronary arteries possibly arising from heparin-induced thrombocytopenia and disseminated intravascular coagulation.[22] Acute MI is also said to result more commonly from non-atheromatous causes in young women than in males.[23]

Of particular interest is our finding of an apparently recent intracoronary thrombosis 2 months after an acute MI; moreover, the coronary arteries appeared normal on angiography approximately 5 months after the initial event. Early interventional coronary arteriography has confirmed the common belief that coronary thrombi tend to disappear fairly rapidly after an acute MI, particularly if there is no clear underlying coronary atherosclerosis. This finding has added to the confusion about the relationship of acute coronary thrombosis to MI.[24,25] Another possible explanation in our case is that the 'coronary thrombus' in the LAD originated in the large mural thrombus situated in the left ventricle and was totally spurious. If this was true then there may have been an acute coronary embolus which caused no further symptoms, possibly because there was an extensive and complete transmural MI subtended by that particular coronary artery. A not too dissimilar clinical situation has previously been documented in mitral valve prolapse.[26] Coronary embolism is generally considered to be a rare event,[27] but some authors believe that it is underestimated clinically and may be most important in assessing pathophysiological mechanisms in patients with MI and normal coronary arteries on angiography.[28] Underlying cardiac valvular disease should suggest a possible complication of coronary embolism,[28] while other associated cardiac conditions are cardiomyopathy, coronary atherosclerosis, and chronic atrial fibrillation.[27] Of relevance to the case under discussion is the finding of mural thrombi in 33% of a large series of patients with coronary artery embolism causing acute MI.[27] The LAD appears to be the vessel most frequently affected; because coronary emboli often lodge distally a small transmural MI often ensues.[29] The large 'recent-looking' coronary thrombus in our patient was quite probably a coronary embolus originating in the left ventricular mural thrombus and of no real clinicopathological importance. The normal appearance of the coronary arteries at the second cardiac catheterization makes this possibility more likely.

Our patient appeared to develop complete RBBB approximately a week after the MI, at which stage he already had features of left anterior hemifascicular heart block (LAHB). Atkins *et al.*[30] in a large series of patients with acute MI found a 43% incidence of complete heart block complicating LAHB and RBBB. This complication was more likely to occur if first-degree atrioventricular block was also present. Atkins *et al.*[30] reported a high incidence of late sudden death in patients with RBBB and LAHB who did not receive a permanent cardiac pacemaker before discharge, while patients with similar peri-infarction conduction abnormalities (such as complete heart block) who were given a pacemaker survived. Atkins *et al.*[30] therefore pleaded for the insertion of a pacemaker if complete heart block followed an MI, even if this conduction defect was intermittent, and recommended insertion of a temporary right ventricular electrode. Similar findings were reported by Waugh *et al.*[31] and by Ritter *et al.*[32] Mullins and Atkins[33] were more uncertain about the efficacy of permanent cardiac pacing in those patients with RBBB and LAHB complicating an acute MI who fail to develop

transient complete heart block. More recently Hindman *et al.*[34,35] agreed with this. My patient did not develop complete heart block and was therefore probably not at such a great risk of sudden death. Therefore, failure to insert a permanent pacemaker will probably make little difference to his prognosis. However, this remains to be verified during follow-up at the Cardiac Clinic.

I wish sincerely to thank Miss H. Weymar of the Cardiac Clinic, Tygerberg Hospital, for preparing the manuscript and the ECGs. Thanks are also due to Mr Chris Wilberforce, Head of the Department of Photography, for his preparation of the photographs. Finally, Dr J. P. van der Westhuyzen, Chief Medical Superintendent of Tygerberg Hospital, is thanked for permission to publish.

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