

# Acute coronary thrombus formation after stress testing following percutaneous transluminal coronary angioplasty

## A case report

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### Summary

Successful percutaneous transluminal coronary angioplasty (PTCA) was performed on a 37-year-old white man with an isolated 95% right coronary artery stenosis who initially presented with type II unstable angina. Submaximal treadmill stress testing was not carried out before PTCA, but testing 3 days after PTCA was strongly positive without accompanying symptoms of myocardial ischaemia. Some 30 minutes after this test the patient experienced severe precordial pain with features of a hyperacute transmural inferior myocardial infarction. Immediate coronary arteriography delineated fresh thrombus related to the previous PTCA site. Intracoronary thrombolysis with streptokinase was successful, revealing an underlying severe stenosis at the PTCA site. PTCA was not repeated, nor was emergency coronary artery bypass grafting (CABG) performed. This is the second such case documented in the literature; the first patient failed to respond to intracoronary thrombolysis with streptokinase and was submitted to emergency CABG. The possible underlying pathophysiological mechanisms are discussed. We believe that the late thrombus formation was directly related to submaximal stress testing after successful PTCA, and recommend that testing to assess the efficacy of PTCA be deferred until at least 1 month after the procedure to allow for completion of the healing process.

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### Case report

A 37-year-old white man was asymptomatic until 5 August 1983, when he experienced severe crushing retrosternal discomfort postprandially. This sensation was unrelated to effort and radiated to the throat with accompanying nausea and

vomiting. It lasted for several minutes, only to be followed in rapid succession by further episodes more severe in intensity and longer in duration. He was transferred to the Intensive Coronary Care Unit (ICCU) at Tygerberg Hospital, Parowvallei, CP, on the same day. The only major risk factor was that he had smoked 40 cigarettes a day for many years. He was otherwise asymptomatic and gave no history of any significant illnesses or operations.

On admission to the ICCU he was not in any particular distress. Examination revealed a normal regular radial pulse with all peripheral pulses easily palpable. The jugular venous pressure was not elevated and the blood pressure was 130/80 mmHg. There was no evidence of any cardiomegaly or congestive cardiac failure. A prominent fourth heart sound was audible but the first and second heart sounds were normal in quality. No murmurs could be heard, but scattered rhonchi were detected. The remainder of the physical examination was negative.

A chest radiograph demonstrated a normal cardiac silhouette and clear lung fields. The resting 12-lead ECG was distinctly abnormal in that inferior ST-T-wave segment changes indicative of myocardial ischaemia were present (Fig. 1, a). Serum cardiac enzyme levels and routine blood biochemical values were normal. A diagnosis of type II unstable angina was made and therapy instituted with oral calcium antagonists,  $\beta$ -blockers, transdermal nitroglycerin (Nitradisc), and anticoagula-

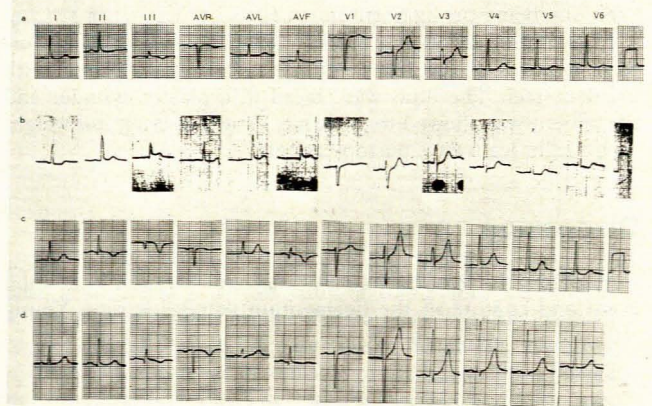


Fig. 1. Resting 12-lead ECGs taken: (a) on admission (full standardization) — markedly flat T waves are seen in the inferior leads with inversion in leads III and aVF; (b) during chest pain 30 minutes after exercise testing — 2 mm ST-segment elevation is seen in leads II, III and aVF, and 1 mm horizontal ST-segment depression is seen anterolaterally (these features are in keeping with a hyperacute inferior myocardial infarction); (c) 3 days after chest pain — characteristics of an evolving acute transmural inferior myocardial infarction are present; and (d) 6 months after PTCA — the tracing demonstrates small Q waves in the inferior leads.

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tion with continuous infusion of heparin 30 000 U/24 h. Within the next 24 hours the patient's chest pain subsided and the resting ECG reverted to normal. However, over the succeeding 5 days he experienced further episodes of chest pain and associated intermittent ECG changes in keeping with non-transmural myocardial ischaemia. During this period the medication was increased to the maximum dosage. Serial cardiac enzyme levels remained within normal limits. On 11 August the patient was submitted to coronary arteriography with a view to coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) if indicated.

### Cardiac catheterization

Selective coronary angiography in the left anterior oblique (LAO) and right anterior oblique (RAO) views demonstrated a normal left coronary artery, but asymmetrical 95% stenosis was noted in the first part of the dominant right coronary artery (RCA) just at the origin of a small right ventricular branch (Fig. 2). The rest of the RCA appeared normal and there was no collateral filling from the left coronary artery. The RCA stenosis was not diminished by sublingual isosorbide dinitrate, nifedipine or intracoronary nitroglycerin, thus excluding coronary vasospasm.

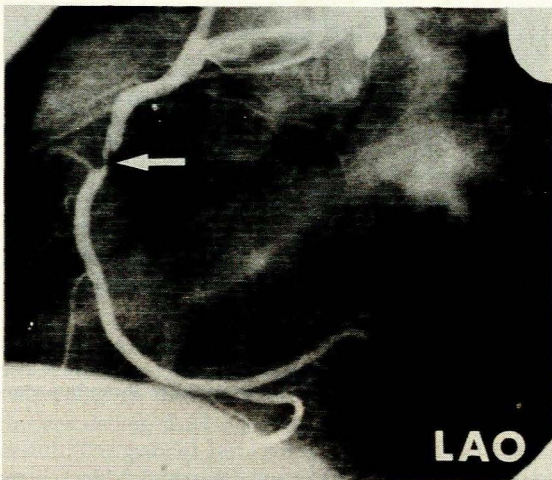


Fig. 2. Right coronary cine angiogram in the LAO view. The vessel is dominant and exhibits a 95% asymmetrical lesion (arrowed) in its first part. The rest of the artery appears free of disease.

In view of the findings of coronary arteriography it was decided to undertake PTCA. Treatment with dipyridamole 100 mg 8-hourly and aspirin 75 mg/d was started immediately; the patient continued to receive nitrates, a calcium antagonist and a  $\beta$ -blocker. The heparin infusion was discontinued on 15 August before undertaking PTCA on 16 August.

### PTCA

This procedure<sup>1</sup> was carried out using a 20 mm long balloon with a diameter of 2,5 mm. The intrinsic gradient was 12 mmHg and the initial gradient across the stenosis 78 mmHg (90 - 12 mmHg). Following the last balloon inflation of 8,5 atmospheres this fell to 38 mmHg. As there was no further reduction in this gradient after repeated inflations at maximum pressure, this residual stenosis (as assessed by the gradient) was accepted; RCA cine angiograms depicted a reduction in the lesion to a 35% stenosis (Fig. 3). The PTCA was therefore considered to be a 'primary success'. The heparin administered

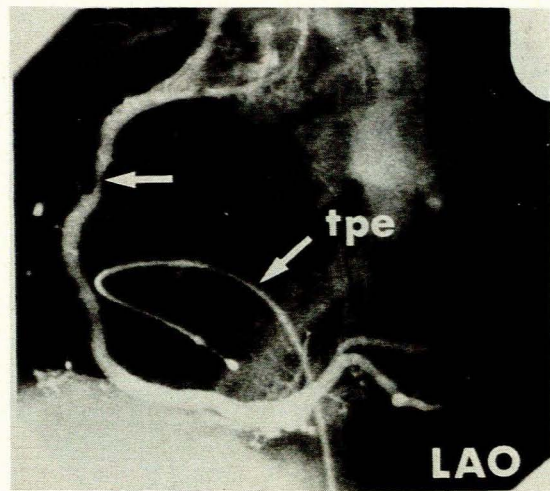


Fig. 3. Selective right coronary cine angiogram in the LAO projection following successful PTCA. A residual 35% stenosis is visualized (arrowed) (tpe = temporary pacemaker electrode in the right ventricle).

during the procedure was electively not continued on return to the ICCU.

The patient was maintained on transdermal nitroglycerin, an oral calcium antagonist and a  $\beta$ -blocker, in addition to dipyridamole and small doses of aspirin. Cardiac serum enzyme values (measured daily) and repeated ECGs remained normal, and there were no further episodes of chest pain. In accordance with the recommended protocol, the patient was therefore mobilized 24 hours after PTCA and a submaximal treadmill exercise test was arranged on 19 August, 3 days after the procedure. The submaximal effort test (85% of predicted maximum heart rate) revealed maximum downward-sloping ST-segment depression of 4,2 mm in the lateral leads immediately after exercise. The degree of ST-segment depression became less pronounced later into the post-exercise recovery phase. The patient had no chest pain during or soon after the procedure and the blood pressure and pulse rate responses were satisfactory. However, some 30 minutes after completing the test he noted severe precordial pain which became progressively worse and was associated with marked nausea. A resting ECG documented some 2 mm ST-segment elevation in the inferior leads with horizontal ST segments laterally (Fig. 1, b). The patient was immediately given intravenous morphine and sublingual nitrates and nifedipine, which resulted in some relief of the pain. A diagnosis of a possible hyperacute inferior myocardial infarction (probably transmural) was made and he was immediately taken to the cardiac catheterization laboratory.

### Catheterization 3 days after PTCA

This procedure showed that the RCA was totally occluded at the angioplasty site, distal to which a thrombus was noted (Fig. 4). Intracoronary thrombolysis with streptokinase infusion commenced; complete lysis of the clot was achieved, but a significant underlying obstruction at the previous PTCA site was revealed (Fig. 5). Several boluses of intracoronary nitroglycerin were injected to exclude vasospasm, but the lesion remained unchanged. PTCA was not attempted.

The patient was again given heparin and was maintained on his other drug therapy. Daily serum enzyme estimations and resting ECGs demonstrated an acute transmural inferior myocardial infarction (Fig. 1, c). There was no further chest pain and the remainder of his hospital course was uncomplicated. He was discharged on 30 August, some 4 weeks after admis-

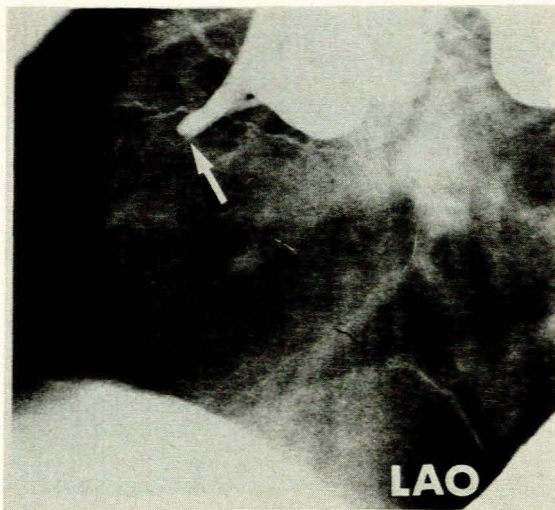


Fig. 4. Right coronary cine angiogram in the LAO projection. Prior to intracoronary streptokinase infusion total occlusion (arrowed) is seen at the previous PTCA site.

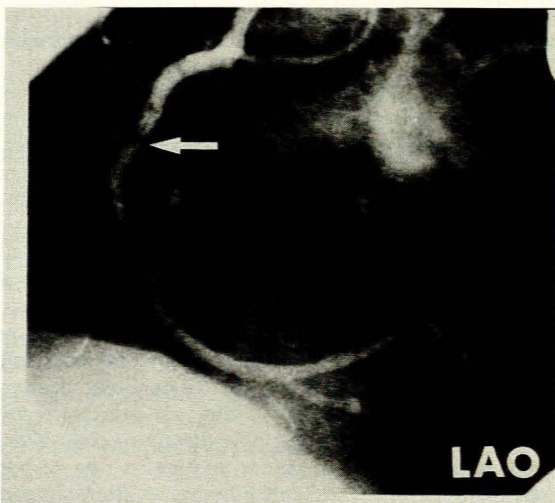


Fig. 5. Selective right coronary cine angiogram in the LAO projection. Following intracoronary streptokinase infusion the coronary circulation is re-established, leaving small thrombi (arrowed) within the lumen proximal to a significant obstruction at the initial PTCA site.

sion, on long-term treatment with a calcium antagonist, a  $\beta$ -blocker, transdermal nitroglycerin, dipyridamole and low-dose aspirin.

**Course following discharge**

On 30 September, 4 weeks after discharge, the patient was subjected to a submaximal stress test (85% of predicted maximum heart rate), which was entirely negative. In addition he underwent a thallium-201 exercise study, achieving his predicted heart rate of 165/min without any angina. This investigation demonstrated decreased isotope uptake in the inferior myocardium immediately after exercise which diminished significantly after 4 hours, features of reversible inferior myocardial ischaemia. The scintiscan failed to delineate previous inferior myocardial infarction.

The patient remained entirely asymptomatic, lost 15 kg in weight, jogged regularly, and stopped smoking. During a second investigation 5 months later he achieved a maximal

heart rate of 152/min over a longer period and again remained free of angina. The isotope distribution immediately and 4 hours after exercise was normal with no evidence of reversible inferior myocardial ischaemia. A submaximal (85% of predicted heart rate) stress ECG failed to demonstrate any ST-T-wave segment abnormalities, and a 12-lead ECG no longer demonstrated transmural inferior myocardial infarction (Fig. 1, d).

**Cardiac catheterization 6 months after PTCA**

This was carried out on 24 February 1984. A left ventricular cine angiogram in the RAO projection delineated a small area of inferior akinesia, the remaining segments contracting normally. Selective coronary angiography again showed a normal left coronary artery, but injection of the RCA demonstrated unanticipated findings. The previous 95% fixed lesion had now dramatically lessened to a 30% fixed obstruction with no evidence of disease in the rest of the coronary artery (Fig. 6).

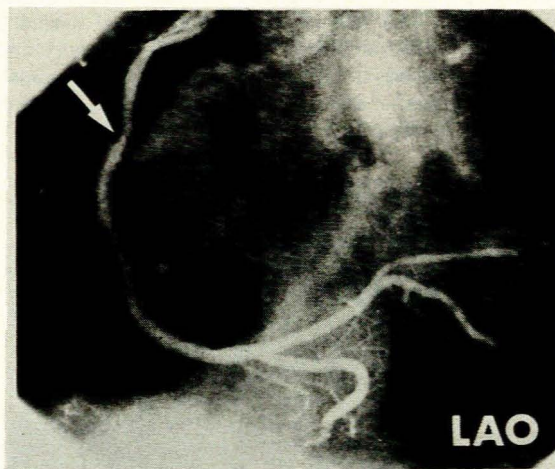


Fig. 6. Right coronary cine angiogram in the LAO projection 6 months after PTCA. A 30% residual stenosis (arrowed) is evident at the site of initial PTCA. No intracoronary thrombus can be seen.

Medication with the calcium antagonist and  $\beta$ -blocker, transdermal nitroglycerin and dipyridamole was discontinued at this stage without any untoward effect. The patient was advised to continue taking a small dose of aspirin and has remained asymptomatic.

**Discussion**

The recent introduction of PTCA in the management of ischaemic heart disease has brought with it complications, the understanding of which may well improve our understanding of the pathophysiological mechanisms responsible for acute myocardial ischaemia and myocardial infarction. For several reasons it has proved an attractive alternative to CABG in patients with significant symptoms. Most clinicians performing PTCA insist on objective documentation of myocardial ischaemia in patients on 'maximum medical therapy' before contemplating PTCA. Submaximal stress (exercise) testing is now routinely used before PTCA.<sup>2</sup> In their initial publication about PTCA, Gruntzig *et al.*<sup>3</sup> recommended submaximal bicycle ergometer effort testing 2 days after PTCA and recently published their results.<sup>4</sup> This time interval seems to have been adopted by most workers. For several reasons we at Tygerberg Hospital have our reservations about this policy.<sup>1</sup> The patient has often been sedentary before PTCA, especially if he was

experiencing unstable angina necessitating several days of hospitalization. We believe that type II unstable angina (the variety with an ischaemic resting ECG or ST-T-wave changes during chest pain and frequent significant underlying obstructive coronary atherosclerosis)<sup>5-7</sup> unresponsive to medical therapy is a contraindication to stress testing because of the very real danger of precipitating an acute myocardial infarction. Our patient therefore did not undergo stress testing before PTCA, although we do undertake this procedure in patients without unstable angina. In the majority of cases the arterial puncture site is at the groin, and patients therefore often complain of significant discomfort which can only be aggravated by stress testing. Furthermore, these patients are all on antiplatelet drugs (such as dipyridamole and aspirin) and some are on anticoagulants (heparin or warfarin), and this medication may cause a most unpleasant haematoma at the puncture site.

Our unwillingness to perform stress testing within the 1st week after PTCA has been further substantiated by more recent publications. Perhaps the most significant is that of Mehta *et al.*,<sup>8</sup> who showed that when subjected to exercise stress testing these patients released thromboxane A<sub>2</sub> (TXA<sub>2</sub>) in amounts far exceeding the release of prostacyclin. The performance of PTCA must of necessity cause some initial disruption, with the ensuing formation of a 'raw area', a situation ideal for platelet aggregation and release of the potent coronary vasoconstrictor TXA<sub>2</sub>. Coronary vasospasm could then well develop.<sup>9</sup> This sequence of events could take place even if the patient was receiving large doses of vasodilator drugs, nitrates, calcium antagonists and antiplatelet agents. The National Heart, Lung and Blood Institute PTCA Registry<sup>10</sup> documented coronary artery spasm in 4.2% of patients during PTCA, and these patients have a higher incidence of coronary artery restenosis (R. K. Myler — personal communication).

Dash<sup>11</sup> recently documented the first and only case of exercise testing-related delayed coronary artery occlusion following successful PTCA. Their patient was a 41-year-old man with unstable angina and a strongly positive exercise test before PTCA. He had an 80% stenosis of the proximal left anterior descending (LAD) coronary artery. PTCA was deemed successful. The patient received treatment with propranolol, nifedipine, oral nitrates and dipyridamole before PTCA, but at no stage was aspirin administered. Heparin and low-molecular-weight dextran were given intravenously during the procedure, as was intravenous and intracoronary nitroglycerin. These drugs were continued after PTCA, but it would seem that heparin was discontinued. Forty-eight hours after the procedure the patient was subjected to a submaximal stress test and reached stage IV of the Bruce protocol without angina. Some 30 minutes later he experienced severe chest pain associated with evidence of acute anterior 'myocardial injury' on the ECG. Coronary arteriography delineated a fresh LAD occlusion at the site of the previous PTCA, which did not resolve despite intravenous and intracoronary administration of streptokinase. The patient underwent emergency CABG and 'subsequently did well'. Dash concluded that the occlusion was probably due to a thrombosis overlying a cracked atherosclerotic plaque and disruption of the intima associated with either dissection of the coronary artery or an occluding intimal flap. He suggested that 'exercise testing in this patient may have contributed to the occurrence of the new coronary occlusion, although the mechanism is uncertain'. It could have increased coronary blood flow and turbulence and possibly caused disruption of the 'reparative process', resulting in haemorrhage and thrombosis. Increased platelet activity<sup>12</sup> was also postulated, as was the fact that the absence of aspirin medication increased the likelihood of thrombosis. Dash con-

cluded that 'exercise testing probably should be delayed more than 48 hours after successful PTCA'.

There are some important differences between the above case and ours. Firstly, we did not perform an exercise test before PTCA, but in the presence of the demonstrated coronary stenosis it is highly unlikely that it would have been negative. Our patient had a strongly positive stress test after PTCA, whereas the stress test of Dash's patient reverted to being negative. Secondly, successful intracoronary thrombolysis with streptokinase in our case revealed significant fixed obstruction at the site of PTCA; his patient failed to respond to this intervention, and the underlying anatomy could therefore not be visualized. Thirdly, his patient received no aspirin, and fourthly, his patient was subjected to emergency CABG whereas we chose to be more conservative after successful intracoronary thrombolysis and in the absence of chest pain with haemodynamic stability. Both these patients did not continue to receive heparin, and this may well have predisposed them to fresh intracoronary thrombus formation.

Acute coronary artery occlusion occurring during PTCA itself or within the first 24 hours has been documented in 4.6% of cases,<sup>10</sup> more commonly in patients with severely stenotic lesions, particularly if these are asymptomatic. Falk<sup>13</sup> demonstrated on autopsy material that if plaque rupture took place in stenoses greater than 90% (determined histologically), the most common resulting lesion was an occlusive thrombus rather than an intimal haemorrhage. This observation is pertinent to our case. Explaining the severe fixed obstruction revealed after successful intracoronary thrombolysis is somewhat difficult unless one postulates plaque rupture induced by the stress test with subsequent thrombus formation. Coronary vasospasm cannot be entirely ignored. This patient was one of our early patients to undergo PTCA, which is why we did not repeat PTCA immediately after successful intracoronary thrombolysis.

We have encountered a patient who developed an acute myocardial infarction within a few hours of apparently successful PTCA; immediate intracoronary thrombolysis was effective, and PTCA was immediately repeated on a significant stenosis at the site of the initial PTCA. That patient's subsequent course was uncomplicated. Similar reports were published by Meltzer *et al.*<sup>14</sup> and Goldberg *et al.*<sup>15</sup>

In cases of acute myocardial infarction it is now our policy to combine PTCA with successful intracoronary thrombolysis if a significant underlying stenotic lesion is revealed. This therapeutic approach, within hours of an acute myocardial infarction, has also been shown to be of benefit by Meyer *et al.*<sup>16,17</sup> and Gold *et al.*<sup>18</sup> McConahay *et al.* have even claimed that PTCA carried out within 8 - 30 days of acute myocardial infarction improves the prognosis.<sup>19</sup> This is not surprising, since DeWood *et al.*<sup>20</sup> have highlighted the fact that significant residual stenosis can act as a nidus for further acute myocardial infarction.

We did not undertake emergency CABG in our patient following successful thrombolysis since there was no definite evidence of coronary artery dissection, the patient's chest pain had disappeared, and he was haemodynamically stable. Kutcher *et al.*<sup>21</sup> and Murphy and co-workers<sup>22</sup> did subject their patients in whom PTCA was unsuccessful to emergency CABG, but did not attempt intracoronary thrombolysis. The latter workers also now utilize the intra-aortic balloon pump,<sup>23</sup> since this reduces infarction size in patients undergoing emergency CABG after failed PTCA. Schofer *et al.*,<sup>24</sup> Mathey and co-workers<sup>25</sup> and Sigwart *et al.*<sup>26</sup> described prevention of acute myocardial infarction following acute coronary artery occlusion within hours of PTCA by employing intracoronary streptokinase prior to emergency CABG.

A particularly interesting aspect for discussion concerns thallium-201 stress scintiscans and repeat selective RCA cine angiograms 6 months after PTCA. At no time could a definite

myocardial infarction (fixed 'cold spot') be demonstrated by the scintiscans. The scan performed 4 weeks after PTCA delineated reversible inferior ischaemia, indicating that the residual RCA stenosis following intracoronary thrombolysis with streptokinase was haemodynamically significant. Furthermore, prompt intervention with streptokinase probably prevented a significant myocardial infarction from occurring. This possibility is supported by the fact that the Q waves in the ECG were no longer abnormal 6 months after the myocardial infarction and only a small area of akinesia was visualized on the left ventricular cine angiogram. The surprising finding of a non-significant RCA stenosis 6 months after PTCA — this stenosis having been most severe following thrombolysis — suggests that plaque rupture took place; coronary vasospasm was quite confidently excluded with vasodilators. This experience further raises the question as to whether repeat PTCA immediately after the successful thrombolysis in this particular patient would have been at all beneficial, and also throws some light on the pathogenesis of coronary occlusion within hours of a 'successful' PTCA. A completely normal thallium-201 stress scintiscan 6 months after PTCA demonstrates restoration of coronary blood flow as verified by the RCA cine angiograms, and the absence of significant myocardial necrosis is probably accounted for by early intracoronary thrombolysis with streptokinase during the hyperacute phase of the transmural myocardial infarction.

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