CASE REPORT

# Primary antiphospholipid syndrome with acute myocardial infarction recanalised by PTCA

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**Abstract**

**A 20 year old man with severe chest pain was hospitalised for acute myocardial infarction. Coronary angiography revealed total obstruction of his right coronary artery, which was successfully recanalised by direct percutaneous transluminal coronary angioplasty (PTCA). There was also diffuse thrombi in the left coronary artery that was not recanalised by perfusion with 3000 U pro-urokinase. Anticoagulant therapy was performed after PTCA. Creatine kinase peaked one day after hospitalisation (4805 U/l). The activated partial thromboplastin time was 62.6 seconds (45%). Plasma anticardiolipin IgG antibodies were high (3.8 and 2.7) in repeated examinations. The PTCA site was patent after three months. Primary antiphospholipid syndrome should be considered as a cause of acute myocardial infarction in young adults, and PTCA with anticoagulant treatment is effective for initial treatment of the syndrome.**

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Keywords: primary antiphospholipid syndrome; acute myocardial infarction; percutaneous transluminal coronary angioplasty

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Antiphospholipid syndrome is a thrombotic disorder characterised by antiphospholipid antibodies. Clinical features are thromboses, thrombocytopenia, and recurrent fetal loss.[1] Patients with antiphospholipid syndrome often exhibit positive lupus anticoagulant activity but they infrequently suffer from the typical systemic lupus erythematosus (SLE) that satisfies diagnostic criteria. Thus, antiphospholipid syndrome without clinical features of SLE is called primary antiphospholipid syndrome.[2] We report a case of primary antiphospholipid syndrome that was initiated by acute myocardial infarction without any other thrombotic disorders.

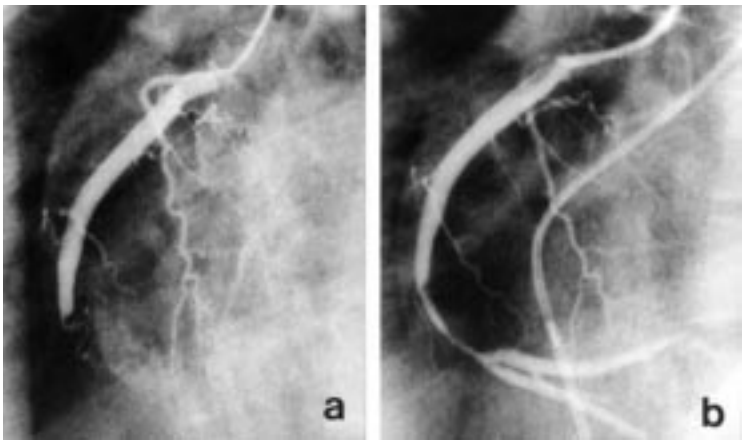


*Figure 1 Right coronary arteriogram on admission shows total obstruction of right coronary artery (a). Arteriogram after PTCA shows successful recanalisation. There is diffuse thrombi in the coronary artery (b).*

## Case report

A 20 year old man with a three month history of chest pain was admitted to our hospital because of severe chest pain and vomiting. He had no risk factors for atherosclerosis (diabetes mellitus, hyperlipidaemia, hypertension). On examination his face was pale, and systolic murmur was audible at the apex. ECG showed ST segment elevation in II, III, aVF, and V6, and ST depression in I, aVL, V5. We diagnosed acute myocardial infarction and performed emergent coronary angiography. The right coronary artery was obstructed totally, therefore, he underwent PTCA with successful recanalisation (fig 1). Although 75% stenosis remained immediately after the PTCA, we finished the PTCA because this stenotic lesion was considered to be caused by thrombi. The left anterior descending artery, diagonal blanches, and septal branch all had diffuse linear defects of contrast medium, suggesting multiple thrombi. A bolus injection of pro-urokinase (3000 U) to the left coronary artery was unsuccessful. Intravenous heparin (15 000 U/day) infusion was performed for the next 10 days, followed by warfarin. Thrombo test was maintained about 25% by oral warfarin (3.5 mg/day). No bleeding complications were noted during thrombolytic and anticoagulation treatment. Serum creatine kinase peaked (4805 U/l) 10 hours after PTCA. Congestive heart failure was controlled by diuretics, nitrate, and low dose dopamine.

The angiography finding of multiple thrombi prompted haematological tests for thrombotic disorders. Blood platelet count was 93 000/ml and prothrombin time was normal (10.3 seconds), but activated partial thromboplastin time was prolonged (62.6 seconds). Anti-DNA antibody was 320 times, but antinuclear

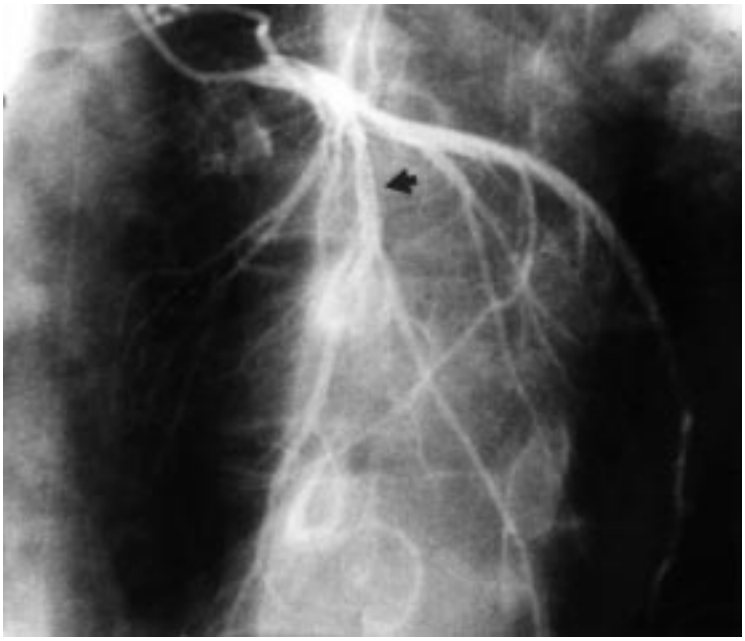

*Figure 2 Left coronary arteriogram after three months shows diffuse organising thrombi in left anterior descending artery (arrows).*

antibody was negative. Lupus anticoagulant was positive, biological syphilis test was negative, and IgG anticardiolipin antibody was 3.8 (normal < 1.0). However, there were no signs nor symptoms of multiple thrombosis or SLE.

Angiography at three months revealed no stenotic lesion or thrombus in right coronary artery. However, in the left anterior descending artery the linear defects suggesting organised thrombi still existed (fig 2). Left ventriculography revealed that the left ventricle was dilated and severely hypokinetic. Left ventricular diastolic internal dimension was 6.78 cm, left ventricular systolic internal dimension 5.02 cm, and echocardiography showed an ejection fraction of 0.50. Lupus anticoagulant was still positive and IgG anticardiolipin antibody was 2.7. Antiphospholipid antibodies tests repeated three months apart remained positive. These findings satisfy the criteria for diagnosis of antiphospholipid syndrome by Harris,[3] and thus he was diagnosed with primary antiphospholipid syndrome because he had no typical signs of SLE.

*Table 1 Case reports of antiphospholipid syndrome with acute myocardial infarction*

| *Case* | *Reference* | *Age* | *Sex* | *Lesion* | *Treatment* | *Other thrombosis* |
|---|---|---|---|---|---|---|
| 1 | Harpaz *et al*[5] | 40 | M | Anterior | t-PA (iv) | Pulmonary embolism amaurosis fugax |
| 2 | Kattwinkel *et al*[6] | 29 | F | Diffuse | Conservative | Not described (recurrent fetal loss) |
| 3 | Thorp *et al*[7] | 29 | F | Inferior | Conservative | DVT |
| 4 | Miller *et al*[8] | 8 | F | Lateral | Resuscitation death | Not described |
| 5 | Ho *et al*[9] | 62 | M | Anterior | t-PA (iv) | DVT |
| 6 | Sakakibara *et al*[10] | 32 | F | Inferior | Conservative CABG | Cerebral infarction |
| 7 | Chambers *et al*[11] | 56 | F | Inferior | Streptokinase (iv) PTCA CABG | Not described |
| 8 | Kovacs *et al*[12] | 56 | F | Diffuse | t-PA (iv) | DVT |
| 9 | Derksen *et al*[13] | 32 | F | Anterior | Conservative | DVT |

t-PA , tissue plasminogen activator; CABG, coronary artery bypass grafting; PTCA, percutaneous transluminal coronary angioplasty; DVT, deep vein thrombosis.

The patient was admitted twice in the following few years because of congestive heart failure but he has not developed further thrombotic disorder.

## Discussion

The most common features of thrombotic disorders in antiphospholipid syndrome are deep vein thrombosis, pulmonary thromboembolism, and stroke. Their occurrence is often multiple and repeated, but this syndrome is rarely initiated in the coronary arteries. According to Asherson *et al*, nine of 13 antiphospholipid syndrome patients with myocardial infarction had previous recurrent deep vein thromboses; only one patient had myocardial infarction before any evidence of vascular occlusion.[4] Table 1 shows reports of cases of antiphospholipid syndrome with acute myocardial infarction since the report by Harris.[5–13] In three of nine patients with antiphospholipid syndrome and myocardial infarction there was no description as to whether there were other thrombotic disorders, except case 2 who experienced four spontaneous fetal losses. One of the mechanisms of fetal loss in antiphospholipid syndrome is suspected infarction of the placenta due to thrombus. Therefore, we cannot confirm that any of these patients have primary antiphospholipid syndrome with myocardial infarction but no other thrombotic disorder.

Widespread cardiac dysfunctions due to multiple arteriolar thrombi are reported in cases of antiphospholipid syndrome even with normal valves and coronary arteries.[14] Case 2 had diffuse ST segment depression and global dysfunction of left ventricle with some segmental heterogeneity in echocardiographic examination. A right ventricular endmyocardial biopsy of this patient revealed multiple small vessel occlusion due to thrombi. The echocardiographic examination of case 8 disclosed that basal segments were normal but mid and apical lesions were akinetic to dyskinetic, suggesting the existence of intramyocardial thrombosis. The left ventriculography of our case revealed a severe diffuse hypokinesia in association with akinesia in the area of myocardial infarction, suggesting the existence of more diffuse intramyocardial thrombi.

The trial of PTCA for the coronary occlusion of antiphospholipid syndrome was unsuccessful (table 1, case 7), and coronary bypass was performed in this case. It seems we are the first to succeed in using direct PTCA for antiphospholipid syndrome with myocardial infarction. We confirmed that the PTCA site was still patent three months later. Anticoagulant therapy commenced immediately after the PTCA may have contributed to such long term coronary patency. In the left coronary artery there were many thrombi so we also injected pro-urokinase (3000 U) as the thrombi were diffuse. Harpaz *et al* and Ho *et al* successfully used intravenous thrombolytic treatments in cases 1 and 5 (table 1).[5 9] Thus thrombolysis may be effective as initial treatment for acute thrombotic disorder including acute myocardial infarction. In our case, we assumed that

thrombi in the left coronary artery had already been organised when emergent angiography was carried out. If thrombi are thought to be organised, intracoronary thrombolysis is ineffective.

Acute myocardial infarction is unusual in young adults, but it has been reported in patients with antiphospholipid syndrome. In conclusion, when young patients with acute myocardial infarction are examined, we should perform immunological tests (lupus anticoagulant, anticardiolipin antibodies, etc) and examination of thromboses of multiple organs. In such a case, PTCA is effective if it is followed by anticoagulant therapy.


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