Myocardial infarction with acute thrombosis of multiple major coronary arteries: a clinical and angiographic observation in four patients

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Acute myocardial infarction; Coronary angiography; Coronary angioplasty.

Four patients are reported in whom concomitant obstructive thrombosis of two major coronary vessels was observed at coronary angiography during evolving myocardial infarction. In all cases the simultaneous involvement of both vessels as the cause of acute ischemia was confirmed by the results of sequential treatment of the lesions with emergency angioplasty.

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Introduction

Acute myocardial infarction (AMI) sustained by acute occlusion of multiple major coronary arteries is a rare clinical observation[1-4]. We describe 4 cases of AMI with double-vessel acute thrombosis treated with emergency coronary angioplasty and stenting in our institution.

Description of cases

Case 1. A 43-year-old male smoker with a history of hypertension and diabetes was admitted to our cardiac catheterization laboratory 2 hours after the onset of typical chest pain. Physical examination showed basal pulmonary rales, a blood pressure of 80/60 mmHg and a heart rate of 100 b/min. The ECG showed antero-lateral AMI, with ST-segment elevation in leads V_1-V_6, I and aVL.

Emergency coronary angiography showed a congenital abnormality, with the origin of both the left anterior descending coronary artery (LAD) and the right coronary artery (RCA) from adjacent ostia in the right sinus of Valsalva; the former vessel was occluded in its middle segment, the latter in its proximal segment (Fig. 1); collaterals from the left circumflex coronary artery (LCx) to the distal RCA were also apparent.

Aspirin 500 mg, heparin (10 000 IU) and abciximab 17 mg bolus were administered intravenously, and abciximab infusion was started. Intra-aortic balloon pumping was also initiated. Both the LAD and the RCA showed spontaneous reperfusion with Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow after the first injections of dye, with residual severe thrombotic lesions in both vessels (Figs. 2 and 3). Chest pain was still present at that time.

We proceeded with coronary angioplasty of the LAD and the RCA, and a single stent was implanted in both vessels (2.75 × 15 mm in the LAD, 3.5 × 18 mm in the RCA), with the sequential restoration of a normal TIMI grade 3 flow in each vessel and a residual stenosis of 5% at both occlusion sites. Only at the end of the procedure did the patient become asymptomatic, and a marked reduction in ST-segment elevation was observed. Ticlopidine 500 mg/die was started the day of the procedure. Peak creatine phosphokinase (CK) was 670 U/l (MB 86 U/l) at 12 hours. The intra-aortic balloon pump was removed after 3 days. Insulin was given to control hyperglycemia. On day 8 the patient suffered from reinfarction, with ST-segment elevation in leads V_1-V_6, I and aVL, complicated by cardiogenic shock and grade III atrioventricular block. Emergency coronary angiography showed subacute thrombosis of both stents, with occlusion of the LAD and

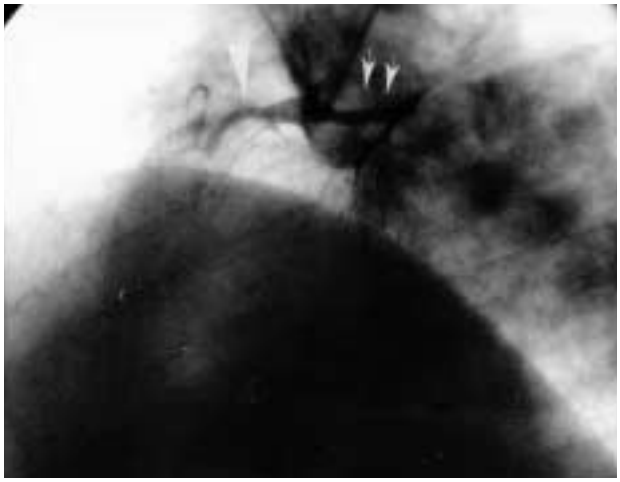

Figure 1. *Case no. 1. Angiogram in the left anterior oblique projection. Both the right coronary artery* (arrow) *and the left anterior descending coronary artery* (double arrow) *arise from a common origin in the right coronary cusp, and are occluded in their proximal segment.*

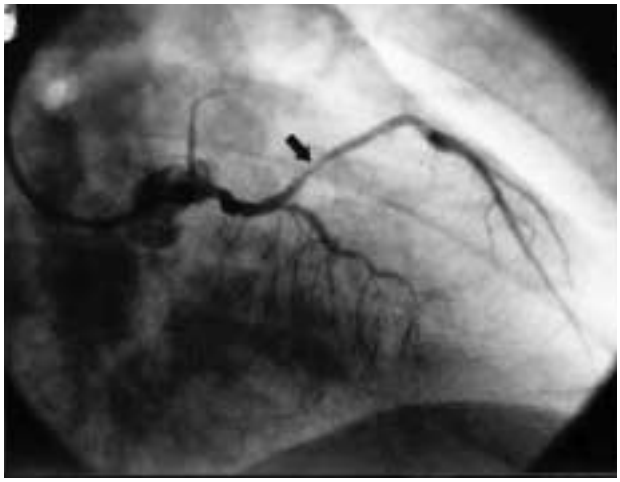

Figure 2. *Case no. 1. Angiogram of the left anterior descending coronary artery in the right anterior oblique projection. Spontaneous reperfusion after the first injections of dye. Residual severe stenosis* (arrow) *at the formerly occluded site.*

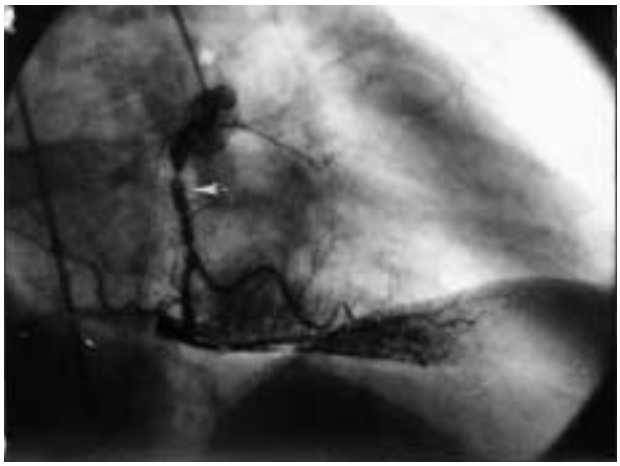

Figure 3. *Case no. 1. Angiogram of the right coronary artery in the right anterior oblique projection. Spontaneous reperfusion after the first injections of dye. Residual severe stenosis with intraluminal thrombus* (arrow) *at the formerly occluded site.*

subocclusion of the RCA, and repeat coronary angioplasty was successfully performed. A new peak of CK at 1482 U/l (MB 144 U/l) was observed at 5 hours. A pre-discharge echocardiogram showed a left ventricular ejection fraction of 0.52. The patient was discharged on day 18. Both the platelet count and plasma fibrinogen concentration were within normal limits at admission, and rose to 507 000/mm^3 and 639 mg/dl, respectively, on day 14. Due to the perceived high thrombotic risk, s.c. low molecular weight heparin was prescribed for 15 days after discharge, together with aspirin and ticlopidine.

At the 6-month follow-up the patient complained of angina Canadian Cardiovascular Society class III and coronary angiography documented severe in-stent restenosis in both vessels. Elective surgical revascularization with left and right mammary arteries was successfully performed.

Case 2. A 70-year-old male smoker, with hypertension, diabetes, previously stable angina for 1 year and a family history of coronary artery disease was admitted to our laboratory 2.5 hours after the onset of typical chest pain. The ECG showed sinus bradycardia (35 b/min), left bundle branch block, and inferior-posterior-lateral AMI, with significant (3 to 6 mm) ST-segment elevation in leads II, III, aVF, V_5-V_6, and ST-segment depression in leads V_1-V_3, aVR and aVL. There were no clinical or ECG signs of right ventricular involvement, while signs of initial left ventricular failure were apparent (Killip class II). Emergency coronary angiography was performed after positioning a stand-by stimulating electrode in the right ventricle. The coronary angiogram showed a balanced coronary system, and diffuse multivessel disease, with a severe lesion in the distal segment of the LAD with normal flow, a severe complex lesion in the proximal RCA, and a severe lesion with thrombus in the mid LCx (Figs. 4 and 5); flow was TIMI grade 2 in both the RCA and the LCx. The patient was given aspirin, heparin 10 000 IU and abciximab 20 mg bolus intravenously.

The LCx was understood to be the infarct-related artery, due to its evident thrombotic lesion with sluggish flow, to its co-dominant distribution being compatible with the observed ST-segment changes, and to the absence of signs of right ventricular involvement. Coronary angioplasty of the mid LCx was performed, with implantation of a 3.5 × 18 mm stent. A TIMI grade 3 flow was restored, with a 7% residual stenosis. Chest pain was markedly reduced at that time, but no regression of ST-segment elevation could be observed in the 4 monitor ECG leads. Therefore, coronary angioplasty of the proximal RCA was performed and a 3 × 13 mm stent was implanted, which restored TIMI grade 3 flow with a 6% residual stenosis. At the end of the procedure on the RCA the patient became asymptomatic, with a

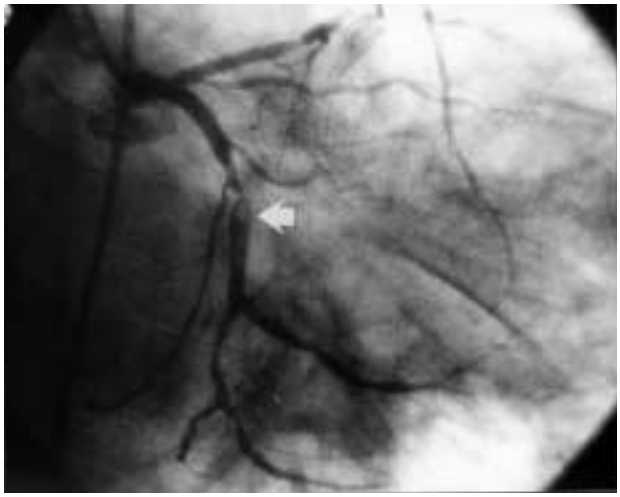

Figure 4. *Case no. 2. Left coronary angiogram in the right anterior oblique projection. Severe stenosis in the mid left circumflex coronary artery with evidence of intraluminal thrombus* (arrow).

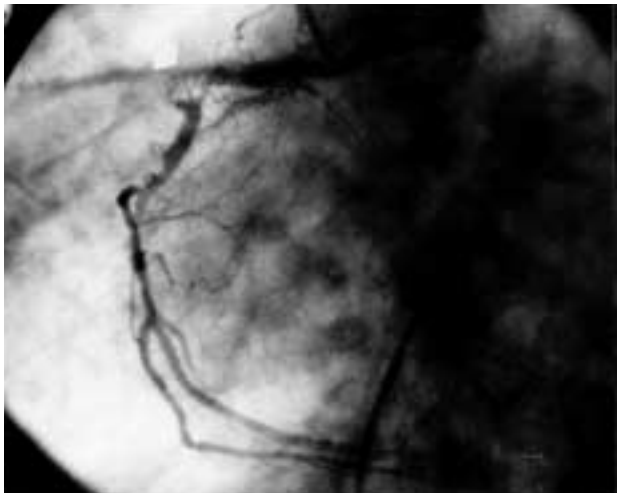

Figure 5. *Case no. 2. Right coronary angiogram in the left anterior oblique projection. Severe complex lesion in the proximal segment* (arrow), *with diffuse involvement of the entire vessel.*

marked reduction in ST-segment elevation and disappearance of all ST-segment depression. The left ventriculogram showed posterior-basal, posterior-lateral, inferior-lateral and diaphragmatic akinesis, with an ejection fraction of 0.29. Ticlopidine 500 mg/die was started the day of the procedure. Peak CK was 14 193 U/l (MB 1322 U/l) at 6 hours. Signs of left ventricular failure persisted during the initial 3 days and required vigorous treatment with diuretics and ACE-inhibitors. Laboratory findings were unremarkable, except for mild hyperglycemia which was controlled by diet. C-reactive protein and fibrinogen levels at admission were 6.34 mg/l (upper normal limit in our laboratory 5 mg/l) and 386 mg/dl, respectively. The patient was discharged uneventfully on day 12 on oral anticoagulants, due to the echocardiographic finding of a possible thrombus at the left ventricular apex.

The patient was admitted 3 months after the procedure due to acute respiratory failure complicated by in-hospital ventricular fibrillation. An automatic cardioverter-defibrillator was implanted. One month later the patient was re-admitted due to subacute recurrent inferior-lateral myocardial infarction complicated by pulmonary edema. One month later the patient was admitted again with cardiogenic shock and post-anoxic coma, with signs that the automatic implantable cardioverter-defibrillator had been triggered by an episode of ventricular fibrillation. He died on day 2 due to refractory left ventricular failure.

Case 3. A 50-year-old man, with a history of non-Q wave inferior AMI was admitted to our laboratory 1 hour after the onset of typical chest pain, with ECG signs of lateral AMI (ST-segment elevation in leads I, aVL, V_5-V_6, and depression in V_1-V_3) (Fig. 6) complicated by multiple episodes of ventricular fibrillation in the Emergency Department treated with DC shock, intravenous lidocaine and beta-blockers. Emergency coronary angiography revealed subocclusion of a small second marginal branch (the likely cause of the previous AMI), occlusion of a proximal large ramus intermedius, and a thrombotic lesion with TIMI grade 2 flow of the proximal co-dominant RCA (Figs. 7 and 8) without any collateral supply. The patient was immediately given aspirin, heparin (8000 IU) and abciximab 21 mg bolus intravenously.

We proceeded with PTCA of the ramus intermedius, and a 3.5 × 18 mm stent was implanted electively, yielding a 3% residual stenosis and TIMI grade 3 flow (Fig. 9). Chest pain, however, did not subside, and the ECG revealed only a partial reduction in ST-segment elevation (Fig. 10). PTCA of the proximal RCA was performed, with direct implantation of a 3.5 × 18 mm stent and a favorable angiographic result. At the end of the RCA procedure the patient became asymptomatic, with almost complete resolution of ECG changes (Fig. 11). Ticlopidine 500 mg/die was started the day of the pro-

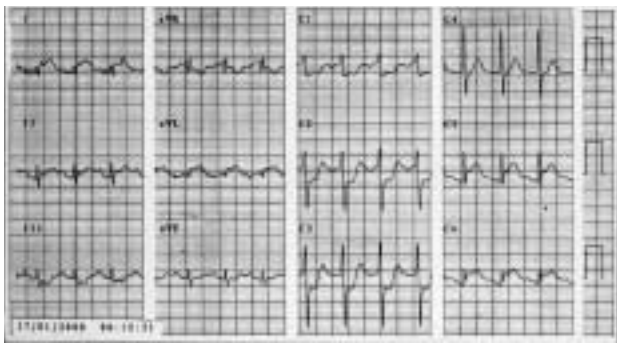

Figure 6. *Case no. 3. ECG at presentation, with signs of acute lateral myocardial infarction (ST-segment elevation in leads I, aVL, V_5-V_6, and ST-segment depression in V_1-V_3).*

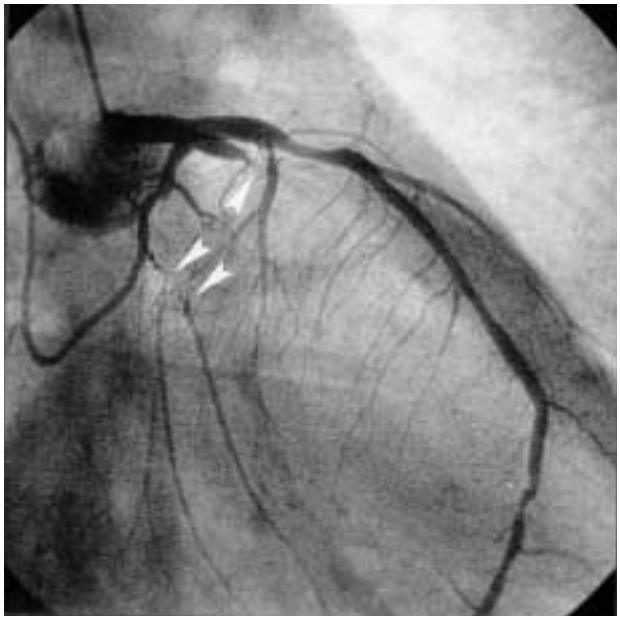

Figure 7. *Case no. 3. Left coronary angiogram in the right anterior oblique projection. A large ramus intermedius is occluded* (arrow). *The second, small, obtuse marginal branch is suboccluded* (double arrow). *A moderate stenosis of the mid left anterior descending coronary artery is also visible.*

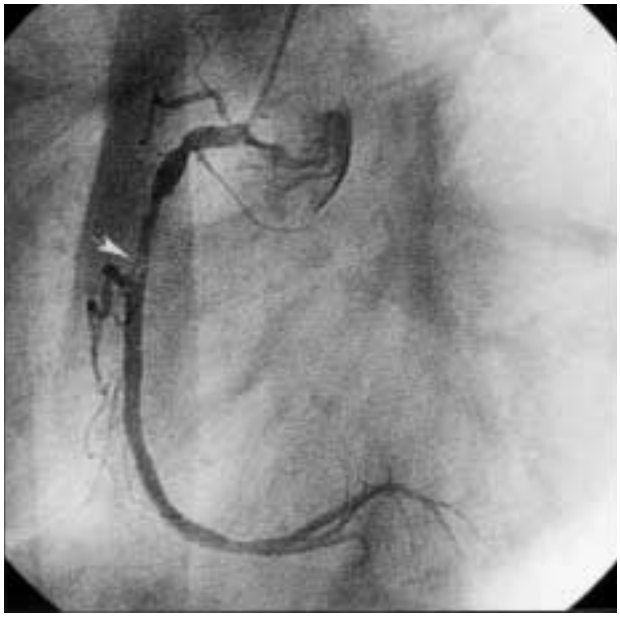

Figure 8. *Case no. 3. Right coronary angiogram in the left anterior oblique projection. A severe stenosis in the proximal segment is visible, with intraluminal thrombus* (arrow).

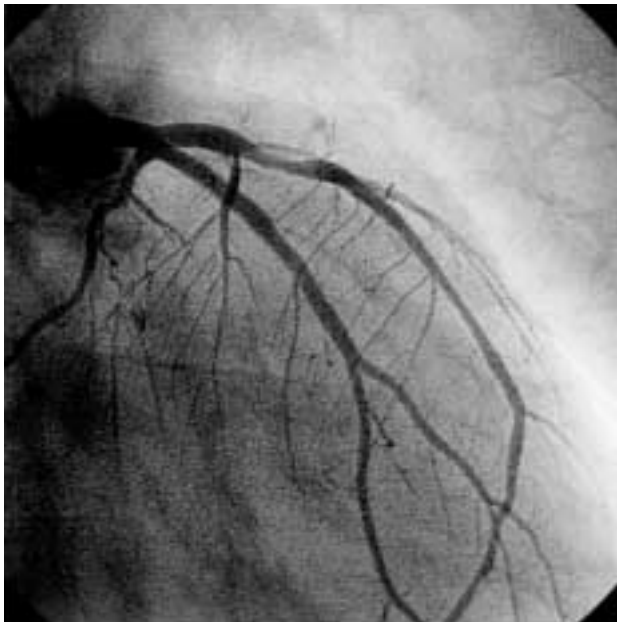

Figure 9. *Case no. 3. Left coronary angiogram in the same projection as figure 7. Result after stenting of the ramus intermedius. The vessel extends to the apex and supplies most of the infero-lateral wall of the left ventricle.*

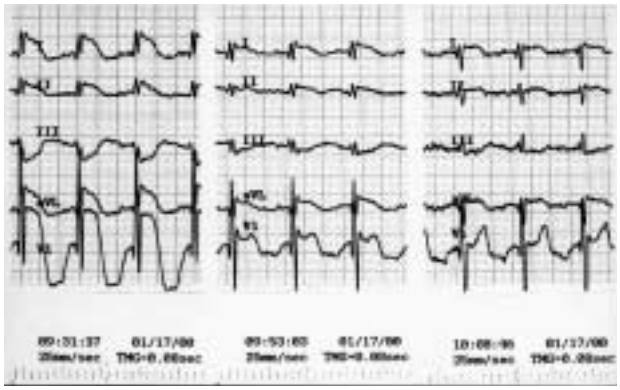

Figure 10. *Case no. 3. Monitor ECG leads before* (left panel) *and after* (mid panel) *reperfusion of the ramus intermedius, and after restoration of normal flow in the right coronary artery* (right panel). *Please note that the tracing marked as V_1 actually corresponds to V_5. Partial resolution of ST-segment elevation is observed in the* mid panel, *which is more marked in the* right panel.

cedure. Peak CK elevation was 14 923 U/l (MB 374 U/l) at 12 hours. All other laboratory findings were unremarkable. The left ventriculogram showed diaphragmatic akinesis and severe apical hypokinesis, with an ejection fraction of 0.35. The patient was discharged uneventfully on day 9 and is symptom-free at 2-month follow-up.

Case 4. A 64-year-old woman, with a history of hypertension was admitted to a nearby hospital in cardiogenic shock 3 hours after the onset of typical chest pain, with ECG signs of inferior and anterior AMI with right ventricular involvement (ST-segment elevation in leads II, III, aVF, V_2-V_5, V3R and V4R). Intravenous lysis with accelerated rt-PA was started while the patient was being transferred to our laboratory. Upon arrival at the cath lab the infusion of rt-PA had almost been completed, chest pain was absent, heart rate was 65 b/min, arterial pressure was 105/70 mmHg, and ST-segment elevation had partly subsided in the precordial leads (Fig. 12). Emergency coronary angiography re-

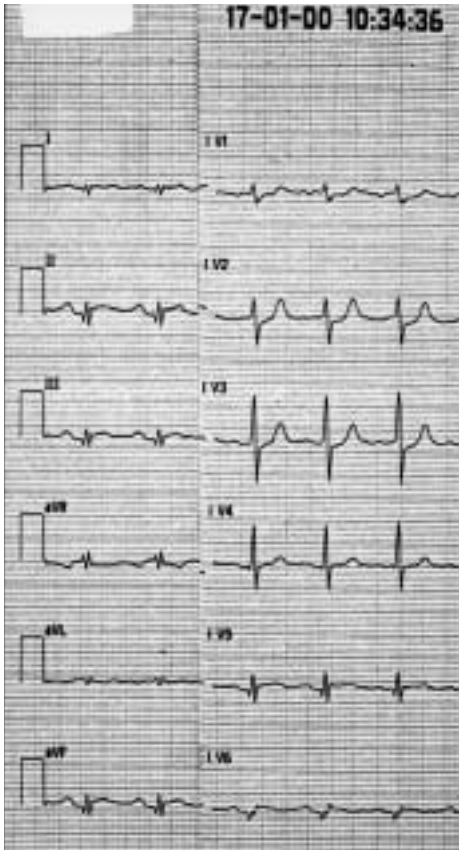

Figure 11. *Case no. 3. Twelve-lead standard ECG after the end of the interventional procedure. Resolution of both ST-segment elevation and depression is now almost complete.*

vealed an occluded proximal RCA (Fig. 13) without collateral supply, and a severe lesion of the proximal LAD with a thrombotic filling defect and TIMI grade 2 flow (Fig. 14). The patient was given aspirin, heparin (3000 IU) and abciximab 10 mg bolus intravenously (two thirds of the standard pro-kilogram bolus).

Direct stenting (3.5 × 13 mm) of the proximal LAD was performed first, yielding a 5% residual stenosis and TIMI grade 3 flow. The ST-segment elevation subsided in both the precordial and the standard leads, and inverted T waves became apparent. Upon new opacifi-

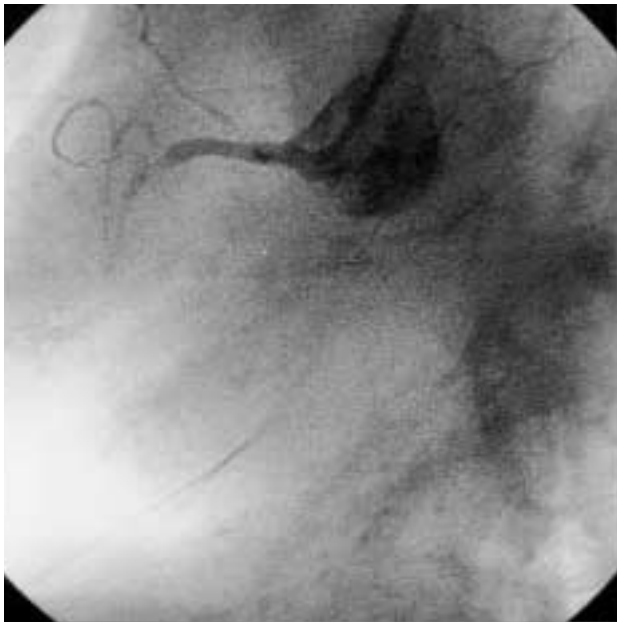

Figure 13. *Right coronary angiogram in the left anterior oblique projection. The proximal right coronary artery is occluded, with a thrombotic aspect.*

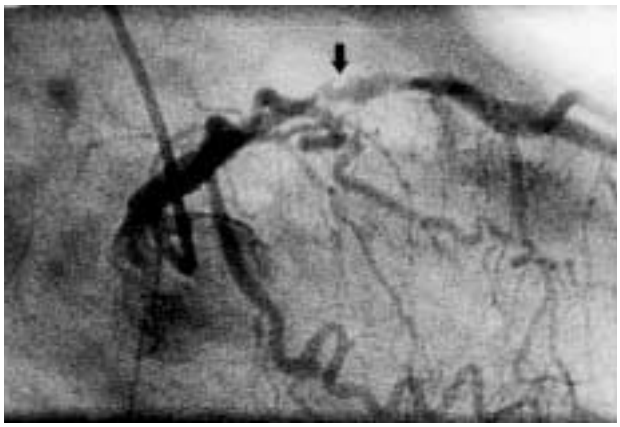

Figure 14. *Left coronary angiogram in the right anterior oblique projection with caudal skew. The proximal left anterior descending coronary artery shows a severe stenosis with thrombotic filling defects* (arrow).

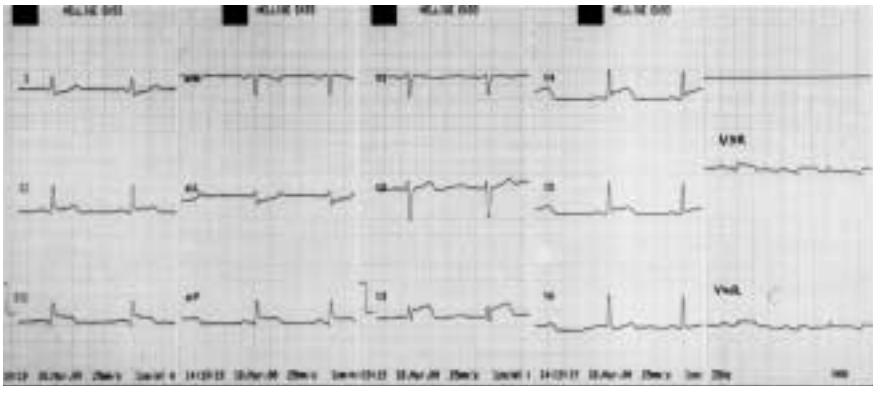

Figure 12. *Case no. 4. Twelve-lead ECG upon arrival at the cath lab. ST-segment elevation is visible in the inferior, left and right precordial leads.*

cation, the proximal RCA was now patent, with a TIMI grade 2 flow, and a single severe proximal stenosis was apparent at the formerly occluded site. Direct implantation of a single 3.5 × 10 mm stent was performed, with restoration of TIMI grade 3 flow and 6% residual stenosis. The intra-aortic balloon pump was inserted at the end of the procedure through the same femoral access; it was removed on day 4. Ticlopidine 500 mg/die was started the day of the procedure. Peak CK elevation was 7500 U/l (MB 760 U/l) at 10 hours. Laboratory findings showed a moderate anemia due to bleeding from the femoral access with no need for transfusion; upon discharge C-reactive protein was 53 mg/l and fibrinogen was 680 mg/dl. Signs of left ventricular failure did not recur. On day 1 the echocardiogram showed severe impairment of the left ventricular function with an ejection fraction of 0.24. It was repeated on day 8: it showed a definite improvement in the kinesis of both the antero-lateral and posterior wall, with an ejection fraction of 0.48. The patient was discharged uneventfully on day 10; she is symptom-free at the 6-month follow-up.

Discussion

Although multiple recent thrombosis has been observed in autopsy studies of patients who deceased for acute myocardial ischemia and in subjects with coronary atherosclerosis who died of non-cardiac causes[5-8], AMI with multiple acute obstruction of major coronary vessels is a very infrequent clinical and angiographic finding.

The contribution of both obstructed coronary vessels to the signs and symptoms of the ongoing acute ischemia was made apparent in all 4 cases by the early angiographic observation of initial occlusion in one or both culprit vessels, or by the concomitant slow flow and the presence of thrombotic lesions in both vessels – as in case no. 2 – and by the evolution of chest pain and ECG changes during and after serial reperfusion with coronary angioplasty.

To our knowledge, only two other cases have been reported in detail in the literature; one was complicated by cardiogenic shock in an adult male, and was treated with primary coronary angioplasty and stenting of both the proximal LAD and proximal RCA which appeared to have acutely occluded[1]; another occurred in a patient with essential thrombocytemia, was caused by subocclusive thrombosis of both the left main coronary artery and the RCA, and was treated with abciximab and emergency coronary angioplasty of the RCA[2]. In addition, one case of concomitant occlusion of both the RCA and LAD in a patient with ECG signs of inferior and anterior AMI and cardiogenic shock was mentioned in a report of direct coronary stenting in AMI[3]. Four cases of AMI related to more than one major vessel were also mentioned in a paper evaluating the efficacy of an invasive strategy in the management of AMI complicated by cardiogenic shock[4]; in three of these the left main stem was involved together with the LAD and/or the LCx, in another patient both the LAD and the RCA were reported to be acutely occluded.

Primary coronary angioplasty is a unique setting where multiple acute coronary occlusion may be observed; it permits both direct visualization of recent thrombotic occlusion of infarct-related arteries, and immediate assessment of the clinical and ECG effects of serial reperfusion of the acutely obstructed vessels. Yet, although reports of primary coronary angioplasty in thousands of patients have been published for more than 10 years, this observation is rare and detailed descriptions of the procedural course and patient outcome are an exception. A possible explanation for this is that AMI with acute obstruction of multiple major vessels causes an extraordinarily extensive myocardial infarction, which usually kills the patient before he can come to medical attention. This is consistent with previous observations in the setting of cardiogenic shock[1,3,4] and with autopsy studies[5-7]. Severe circulatory impairment was also present in 2 out of 4 patients in the present paper. Patients with AMI sustained by acute occlusion of multiple coronary vessels would, therefore, be likely to contribute substantially to the large number of coronary deaths occurring before hospital admission[9].

Acute multivessel obstruction, however, may also be the underlying cause of a “large” AMI in an unknown number of patients who do reach the hospital. In fact, the ECG changes in our patients no. 1 and no. 3 were not so extraordinarily extensive as to suggest multivessel obstruction. The “limited” extension of ECG changes in these 2 patients may be explained by their initial angiographic findings showing some preserved flow in the acutely ischemic territory. Both the LAD and the RCA were initially occluded in patient no. 1, but collaterals were visible from the LCx to the RCA and the ECG showed a large anterior-lateral AMI. Similarly, the ramus intermedius was initially occluded in patient no. 3 but the RCA was patent, albeit with sluggish TIMI grade 2 flow, and the ECG showed a lateral AMI.

Therefore, our observation suggests that acute occlusion of multiple major coronary vessels may occur more often than expected in patients admitted with AMI, and also in patients without ECG signs of a specially large AMI and/or not presenting with cardiogenic shock.

Our findings may support the hypothesis that ST-segment elevation AMI, as well as non-ST-segment elevation acute coronary syndromes, may be associated with a systemic acute pro-thrombotic condition, possibly involving inflammatory mechanisms as well[10,11], which are not confined to a single spot in the coronary circulation. A greater proportion of non-culprit coronary lesions with rapid angiographic progression or regression in patients with AMI vs stable angina has been reported[12], suggesting that AMI may be the hallmark of

a systemic coronary disease activity. High catecholamine levels can increase platelet activation and thrombin generation, can participate in triggering AMI[13], and might be responsible for thrombotic occlusion at the site of multiple "vulnerable" plaques. Although changes in C-reactive protein and other markers of inflammation were not assessed during hospital stay in our patients, C-reactive protein levels were slightly elevated in one of them on admission, and became elevated at discharge in another; two other patients developed high fibrinogen levels and/or elevated platelet counts during hospital stay. Subacute occlusion of both stents in one of these 2 patients despite heavy anti-thrombotic medication may be consistent with a systemic thrombotic and/or inflammatory reaction, possibly enhanced by the initial percutaneous intervention[14].

Spontaneous dissection of coronary plaques should also be considered. It was observed in 1% of cases in a prospective study of 3803 consecutive first angiographic examinations, and was most often associated with acute coronary syndromes[15]. Cases involving multiple major vessels have been the object of both clinical[16-20] and autopsy[21] reports. Although small coronary dissections may escape visualization at angiography, no signs of dissection were apparent on the angiograms of our patients upon careful review by several experienced operators.

A different causative explanation is likely in at least 2 of our patients, i.e. acute occlusion/subocclusion of one large culprit vessel may have triggered a condition of reduced cardiac output, thereby compromising flow in another coronary vessel where a severe "silent" stenosis was already present, and causing acute "secondary" thrombosis. The observations in 2 of our patients, and in others with cardiogenic shock[1,3,4], albeit compatible with the hypothesis of a "low-flow" driven thrombosis of a second vessel, cannot provide any definite answer as to whether the shock was a cause for, or an effect of, acute occlusion of multiple coronary vessels. An acute adrenergic response secondary to initial occlusion of one coronary vessel and subsequent pump failure might have played a role also in this setting, by causing or favoring thrombosis at the site of a second preexisting vulnerable plaque.

Although intravenous lysis with rt-PA appeared to be of help in the only patient (no. 4) in whom it was used in this series, multivessel acute thrombosis may be a specially difficult setting for reperfusion by intravenous lytics, especially in the presence of low cardiac output. If intravenous lytics are administered, proof of multivessel involvement, however, is definitely difficult to obtain – except with acute angiography – in these patients when the ECG findings are not so clear-cut as in case no. 4. In fact, the effects of reperfusion of one or both acutely obstructed vessels may be difficult to assess on clinical or ECG examination alone, and delayed coronary angiography may not prove that occlusion was recent, even when it is complete and persistent, and angiographic images no longer show non-occlusive intraluminal thrombus.

In conclusion, patients with multivessel acute thrombosis in AMI seem to be a very high-risk and poorly recognized subset. In these patients acute coronary angiography is the only way this condition can be detected with certainty in life, and emergency coronary angioplasty, possibly associated with the use of potent anti-thrombotic and/or lytic drugs, may be the best available treatment to achieve quick reperfusion. The causes of acute occlusion of multiple coronary vessels remain elusive. Study of these patients may provide insight into some of the mechanisms of acute vessel thrombosis in AMI.

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