Dynamic left ventricular outflow tract obstruction: an unusual mechanism mimicking anterior myocardial infarction with cardiogenic shock

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Cardiogenic shock is a frequent and threatening complication in the course of acute myocardial infarction. Besides the well known causes (left ventricular failure, acquired interventricular defect, papillary muscle rupture, free wall rupture) other less frequent mechanisms recognize a functional substrate. The recognition of such mechanisms makes us to revert to the treatments with completely different prognostic implications. In our Coronary Care Unit we encountered, in a period of 12 months, 4 patients who presented clinical, electrocardiographic and/or echographic signs and symptoms of acute myocardial infarction, with different degrees of heart failure up to cardiogenic shock. Only 1 patient showed a severe stenosis of the left anterior descending coronary artery and a significant creatine kinase reduction. Left ventriculography, performed at admission, was unable to disclose the true mechanism of clinical presentation. Only a thorough echographic examination disclosed the presence of a dynamic left ventricular outflow tract obstruction as the cause of heart failure culminating in cardiogenic shock. Once recognized, pathophysiological treatment (administration of beta-blockers and withdrawal of vasodilators, inotropic drugs and intra-aortic balloon pump) led to a dramatic improvement, with an almost complete left ventricular function recovery. Left ventricular outflow tract obstruction is a mechanism that can lead to severe heart failure as a complication of an acute myocardial infarction. Conversely such a mechanism can be precipitated by other causes (hypotension, hypovolemia, especially in hypertensive patients) and can mimic an acute myocardial infarction. Its incidence is not negligible: in our Coronary Care Unit it accounted for about 15% of all cases of myocardial infarction requiring inotropic support. An accurate echocardiographic examination is mandatory even after coronary angiography, and always permits the physician to select the appropriate therapy.

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Introduction

Cardiogenic shock is a threatening and not rare complication of acute myocardial infarction (AMI). The most frequent causes leading to cardiogenic shock are left ventricular failure, interventricular septal rupture, left ventricular free wall rupture, and severe mitral regurgitation (MR). The last of the above causes recognizes several different mechanisms: acute ischemic papillary muscle dysfunction is the leading cause (probably due to terminal perfusion of the muscle), left ventricular dilation and chordae tendinae or papillary muscle rupture are other mechanisms. General principles for the treatment of cardiogenic shock include inotropic stimulation, pre and afterload reductions.

Other less known causes of cardiogenic shock during evolving AMI recognize a functional mechanism whose identification leads to different treatments. Echocardiographic examination plays a major role in identifying such patients.

We describe four cases of patients admitted to our Coronary Care Unit over a 12 month period, with evolving AMI early complicated by left ventricular failure ranging from hypotension and mild pulmonary congestion to overt shock. Although coronary angiograms and left ventriculography had been performed in 3 patients out of 4, until an echocardiographic examination, the mechanism of shock remained unexplained and the therapy was unsuccessful. In all patients the recognition of the exact mechanism sustaining cardiogenic shock brought about a change in the treatment with complete resolution of acute cardiac failure and completely different prognostic implications.

Description of cases

Case 1. A 75-year-old woman with a history of mild hypertension was transferred to our Department of Cardiology from a primary hospital in the first hour of an anterior AMI for primary coronary angioplasty (PTCA) because of right hip replacement the day before. On arrival to the cardiac catheterization laboratory chest pain had already diminished, blood pressure was 100/80 mmHg, heart rate 90 b/min; a grade 2/6 holosystolic apical murmur, and fine rales at pulmonary bases were detected. The electrocardiogram (ECG) showed right bundle branch block with ST segment elevation in leads I, II, aVF, and V_3 to V_6 (Fig. 1). A moderate post-surgical anemia (Hb 8.0 g/dl) was present.

The treatment before admission to our Department included atenolol 5 mg i.v., aspirin 325 mg, i.v. nitrates, and transfusion of 2 red blood cell units.

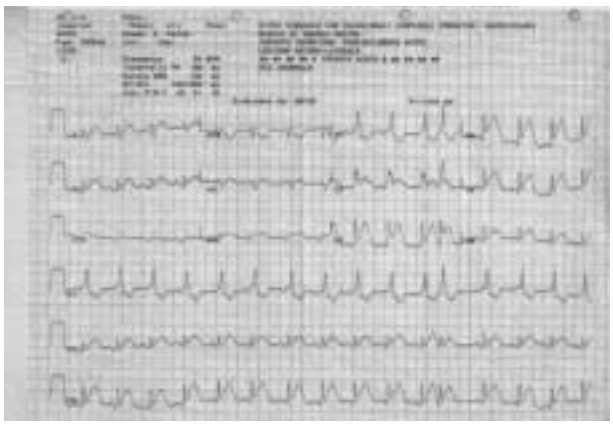


Figure 1. *Case 1. ECG on admission.*

Coronary angiography showed a severe stenosis (95%) at the mid level of the left anterior descending coronary artery (culprit lesion) with restored TIMI 3 blood flow. Minor (up to 40%) stenosis was present on the circumflex branch and right coronary artery. Left ventriculography (Fig. 2) showed a large dyskinesis of the apical wall and a marked hypercontractility of the other left ventricular walls. Ejection fraction was 56%. A third degree MR up to the pulmonary veins was present with inconstant findings of partial posterior mitral leaflet prolapse above the mitral plane. PTCA was not performed because of spontaneous recanalization of the infarct-related coronary artery and the high risk of bleeding complication related to the very recent non-cardiac surgery.

An echocardiographic study showed an enlarged, mildly dyskinetic apex, akinesis of mid-part of the anterior interventricular septum (IVS), anterior and lateral walls, with hyperkinesis of the remaining segments. Wall thickness was uniformly increased (1.3-1.5 cm) with a sigmoid shape of the basal anterior IVS. The left atrium and right chambers showed normal dimensions. A systolic anterior movement of the anterior mitral leaflet and chordae was seen (Fig. 3). Color Doppler flow mapping showed an early systolic flow acceleration in the left ventricular outflow tract (LVOT) followed by a large mid-systolic mitral regurgitant jet directed towards the atrial septum up to the atrial roof). Continuous wave Doppler confirmed the presence of LVOT acceleration with a scimitar shape (measured velocity of 3 m/s) due to a dynamic stenosis created between the anterior mitral leaflet and basal IVS (Fig. 4) and revealed that the mitral regurgitant jet started at mid-systole, after the dynamic gradient had peaked. Pulmonary systolic pressure was 50 mmHg.

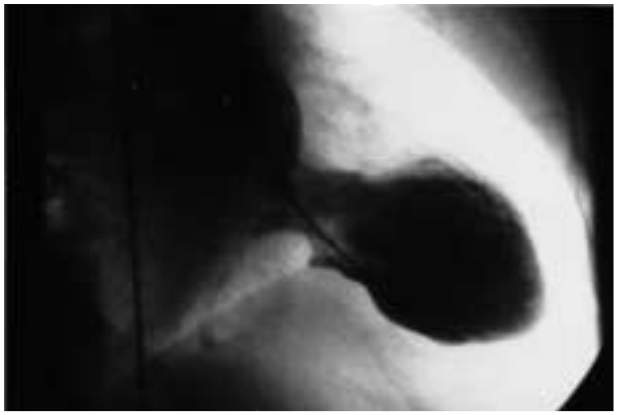


Figure 2. *Case 1. Left ventriculography (right anterior oblique 30°): systolic large dyskinesis of the apical wall and marked compensatory hypercontractility of anterobasal, part of anterolateral, inferobasal and part of diaphragmatic segments. Global ejection fraction 56%. Mitral regurgitation is represented by the dark area behind the valvular plane (grade III).*

Assuming that MR was due to the hyperdynamic state of part of the left ventricle causing mitral apparatus distortion, vasodilators (nitroglycerin) were stopped

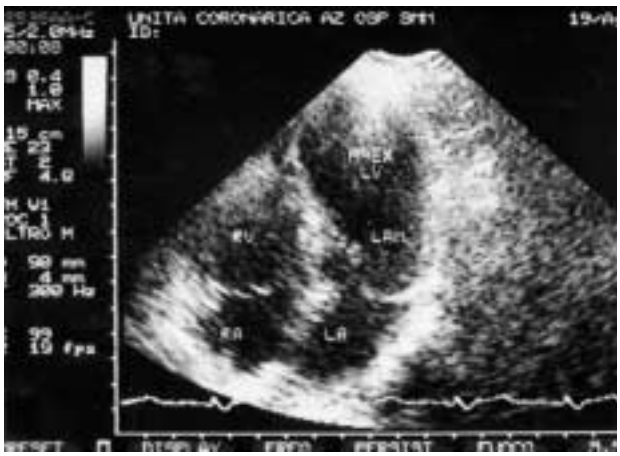


Figure 3. *Case 1. Echocardiographic two-dimensional apical 4-chamber view (mid-systole): dilated apex of the left ventricle (apex LV), mitral anterior leaflet (LAM), and chordae displacement towards the basal septum (hypertrophic). LA = left atrium; RA = right atrium; RV = right ventricle.*

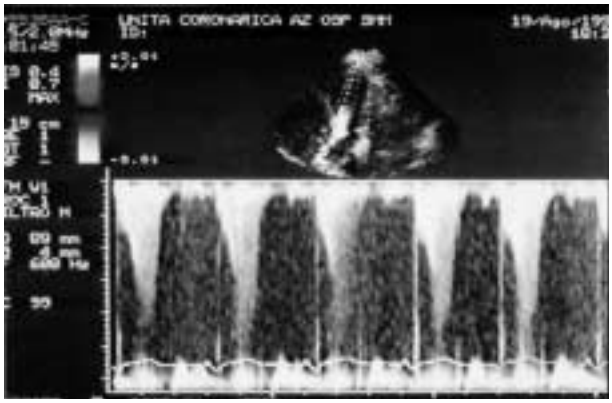


Figure 4. *Case 1. Echocardiography. Continuous wave Doppler discloses the presence of high flow acceleration (3 m/s) due to a dynamic stenosis (scimitar shape) followed by a mid-systolic flow due to mitral regurgitation starting after the dynamic gradient had peaked.*

and beta-blockers started (atenolol 5 mg i.v.). An echocardiographic study 1 hour later remained unchanged apart from a reduction in pulmonary systolic pressure (37 mmHg). Clinical status was stable. Low molecular weight heparin, oral atenolol (50 mg), and ramipril (2.5 mg) were started, 500 ml saline infusion and 2 more red blood cell units were infused over the next 6 hours. Subsequently, despite persistent systolic anterior movement, LVOT flow velocity had reduced (1.3 m/s) and mitral insufficiency was mild at color Doppler; pulmonary systolic pressure and infarct area extension were unchanged. The ensuing clinical course was uncomplicated. Therapy consisted of atenolol 50 mg bid, ramipril 2.5 mg bid, acetylsalicylic acid 160 mg/die, enoxaparin 4000 IU bid. Creatine kinase (CK) peak was 743 U/l (CK-MB 37 U/l) at 14 hours. Deep anterior negative T waves and no Q wave appeared on ECG. On day 6 the akinetic area was limited to the apex and part of the anterior and lateral medium segments; mitral systolic anterior movement disappeared and no MR was detectable; LVOT flow velocity decreased to 1 m/s. In order to evaluate myocardial viability a low dose dobutamine was administered. At 10 μg/kg/min we observed an improvement in contractility of akinetic medium segments of the anterior and lateral wall and mitral systolic anterior movement without regurgitation; at 20 μg/kg/min moderate MR developed and part of the anterior septum, anterior and lateral wall at the medium level became akinetic again. The following day the patient was transferred to the referral hospital from which was discharged 10 days later without further events. At 6 month follow-up the patient was asymptomatic and no major cardiac events were recorded. The echocardiographic examination revealed a normal left ventricular function with a mild apex hypokinesia; hyperdynamic status and systolic anterior movement were absent.

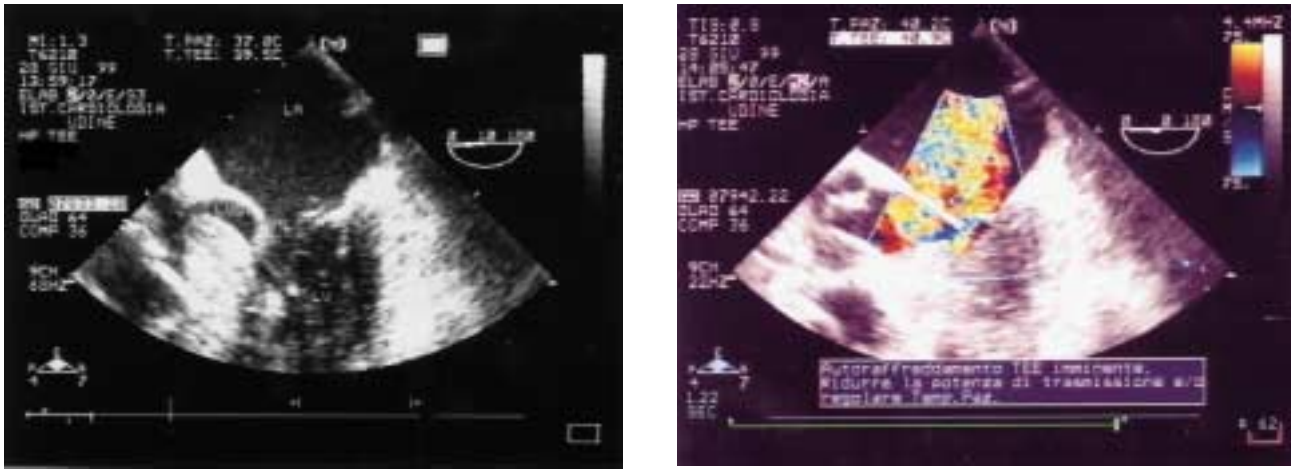
Case 2. A 69-year-old woman with a history of borderline hypertension, smoking, and traumatic rib fracture dated 20 days before, was admitted to a secondary hospital complaining of a heavy substernal chest pain and cardiogenic shock.

ECG recorded in the ambulance showed ST segment elevation in leads I, aVL and V_2 and ST segment depression in III and aVF. Sublingual nitroglycerin was administered with slow resolution of symptoms. On admission the patient was asymptomatic. ECG showed normalization of ST segment elevation with Q wave in V_1 and V_2. Blood pressure was 100/70 mmHg. She was treated with aspirin 325 mg, metoprolol 50 mg and i.v. heparin. During the following hours, in the absence of signs or symptoms of new ongoing ischemia, cardiogenic shock developed with blood pressure falling to 50/30 mmHg. An echocardiographic study showed a large akinetic area of the left ventricle involving the IVS, anterior apex and lateral segments with moderate ventricular dysfunction, and severe MR. Dopamine infusion was started. Mechanical intubation was initiated because of respiratory insufficiency and the patient was transferred to our Department for urgent cardiac catheterization.

Coronary angiography showed normal coronary arteries with a slow flow. Left ventriculography showed a large apical dyskinesis and akinesis of the anterior and lateral segments. Ejection fraction was 35%. Severe MR was also present. Intra-aortic balloon counterpulsation (IABP) was instituted.

The patient was admitted to the General Intensive Care Unit and treated with adrenaline, dobutamine, nitrates, heparin and furosemide. During the following days the patient's clinical status slightly improved, but severe unexplained MR still dominated. Systemic systolic blood pressure rose to 95-110 mmHg, cardiac index was 2-2.5 l/min*m^2, and mean capillary wedge pressure was 30 mmHg. Chest X-ray revealed persistent pulmonary congestion. No attempt at respiratory weaning was made. Surprisingly her total CK level peaked at only 492 U/l, with MB 96 U/l. The cardiac surgeon was consulted in order to evaluate the possibility of mitral valve replacement.

A transesophageal echocardiography performed on day 6 showed that mitral incompetence was due to a systolic anterior displacement of the anterior mitral leaflet with loss of leaflet coaptation (Fig. 5). Hyperdynamic contractility of basal left ventricular segments caused the systolic anterior movement and LVOT obstruction, with a late peaking jet with a measured velocity of 4.5 m/s corresponding to a peak gradient of 81 mmHg. The immediate suspension of inotropic drugs (dobutamine), vasodilators (nitrates) and IABP and the administration of i.v. atenolol bolus, immediately led to a major decrease in regurgitation with a subsequent fall in pulmonary wedge pressure to 22 mmHg, a rise in systemic pressure from 80/40 to 110/60 mmHg and an increase in left ventricular stroke volume (from 24 to 43 ml). Interestingly, when IABP was set at 1:8 inflation/deflation ratio, only the beat following balloon deflation showed anterior mitral leaflet systolic movement and severe MR



A B

Figure 5. *Case 2.* A: *transesophageal echocardiography (5-chamber horizontal view) on day 6. Systolic wide loss of coaptation of mitral leaflets due to systolic anterior displacement of the anterior mitral leaflet (AML).* B: *transesophageal echocardiography (5-chamber horizontal view) on day 6. Color Doppler flow mapping of large mitral regurgitation. LVOT = left ventricular outflow tract. Other abbreviations as in figure 3.*

(Fig. 6). This finding further clarified the pathophysiological mechanism of MR in this patient. Beta-blocker therapy was continued, and IABP removed.

The day after the patient was weaned from mechanical ventilation and transferred to the referral hospital. At 12 month follow-up the patient was stable. Two-dimensional echo showed a completely normal left ventricular function.

Case 3. A few days after the presentation of case 2, a 64-year-old woman with a history of hypertension was urgently transferred to our Department for an anterior AMI with cardiogenic shock. Her symptoms had started 4 hours before. The patient was on mechanical ventilation. Coronary angiography showed only irregularities of the left anterior descending coronary artery. Left ventriculography showed apical aneurysm and hyperdynamic basal segments; an intraventricular gradient of 35-40 mmHg was detected at withdrawal. Ejection fraction was 35%.

Clinical suspicion of a dynamic obstruction was confirmed by an echocardiographic study which measured 70 mmHg intraventricular systolic gradient, due to the presence of a hypertrophied sigmoid basal septum, an anterior mitral leaflet systolic movement with severe meso-end-systolic MR.

I.v. inotropic drugs and i.v. nitrates were stopped and i.v. beta-blocker therapy and crystalloid solution were started. A progressive clinical improvement was soon observed. Her total CK release was 347 U/l (MB 62 U/l).

Two days later echo revealed an ejection fraction of 50%, apical akinesis and mild hypokinesis of anterior septal and anterior medium segments. Intraventricular gradient almost disappeared (8 mmHg) as well as mitral insufficiency (mild). Six days later the patient was discharged with an almost complete recovery of the left ventricle.

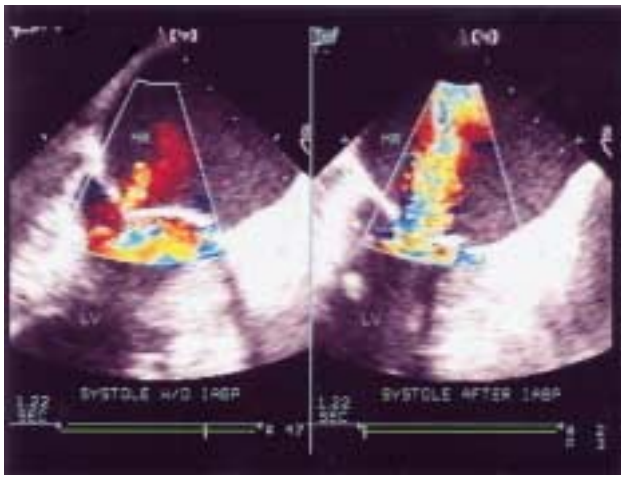


Figure 6. *Case 2. Transesophageal echocardiography (2-chamber vertical view) on day 6. Color Doppler flow mapping of mitral regurgitation (MR).* On the left: *MR without intra-aortic balloon pump.* On the right: *MR following intra-aortic balloon deflation. LV = left ventricle.*

Case 4. A 74-year-old man, with a history of chronic uremia on a hypertensive basis, treated with hemodialysis for 3 years, was admitted to our Coronary Care Unit because of dizziness and dyspnea without chest pain, during dialysis. An ECG was performed with evidence of 2-3 mm ST segment elevation in V_4-V_6, D1, and aVL leads. On arrival he was asymptomatic, blood pressure was 95/70 mmHg, heart rate was 98 b/min he had no decompensation signs, and neurological examination was negative. On cardiac auscultation a 4/6 harsh murmur was audible at the aortic site. I.v. nitrates, aspirin and low doses of i.v. beta-blockers were initiated. The day after the patient was still symptom-free, ECG had "improved" with a reduction in ST segment elevation; systemic arterial pressure was 95/50 mmHg and heart rate 80 b/min. A maximum CK rise of 226 U/l (MB 41 U/l) was recorded.

The cardiac echographic examination showed a small and hypertrophic left ventricle (IVS 13 mm, posterior wall 13 mm) with thinning and dyskinesis of apical segments, hypokinesis of anterior and lateral segments at medium level; a compensatory hyperkinesis of basal segments was present. A mid-ventricular obstruction occurred during meso-systole with a Doppler gradient of 182 mmHg.

Nitrates were stopped. Further beta-blocker and crystalloid solution were given with a progressive increase in systemic blood pressure. The day after echo revealed a complete recovery of apical and anterior segments, with a global ejection fraction of 72%. A mid-ventricular gradient of 100 mmHg was still present.

The ensuing dialytic treatments were administered with a reduced volume depletion and no complication occurred. ECG evolved with ischemic anterolateral T waves.

Over the following days the patient underwent coronary angiography, showing a proximal subocclusive stenosis, with thrombus, of the right coronary artery, critical stenosis of the second diagonal branch, and subcritical stenosis of left anterior descending coronary artery and proximal circumflex artery. Left ventriculography was normal. PTCA with stenting of the proximal right coronary artery was successfully performed. The patient was discharged 4 days after without further complications.

Discussion

Dynamic LVOT obstruction is well described in hypertrophic cardiomyopathies. In addition, other clinical conditions are associated with dynamic LVOT obstruction, anatomical outflow malformation[1-3], as well as with functional states in the presence of predisposing factors[4]. LVOT obstruction has been described in left ventricular hypertrophy[5-9], excessive sympathetic stimulation[10], during dobutamine stress echocardiography[11,12], after aortic valve replacement, after mitral valve surgery or mitral annulus replacement with prosthetic ring[2,13-21], and during cardiac tamponade[22].

Dynamic LVOT obstruction has also been described in the setting of anterior AMI, and different mechanisms have been advocated. Clinical signs of dynamic subaortic stenosis similar to those reported in idiopathic hypertrophic subaortic stenosis were first observed in a patient with AMI and acute hypovolemia (diarrhea), without asymmetric septal hypertrophy at *post-mortem* examination[23]. Furthermore, apical infarction due to mid-ventricular hyperkinetic obstruction without acute coronary thrombosis was observed in hypertensive patients during pre or afterload reduction (e.g. dehydration)[24,25]. The recognition of mitral systolic anterior movement as a possible cause of LVOT obstruction during AMI was reported by Ohtani et al.[26] only in 1994. Other authors have reported mitral systolic anterior movement related to coronary artery occlusion, and its disappearance after successful PTCA[16].

The 4 patients herein described all had clinical signs and symptoms of a large AMI with various degrees of left ventricular failure. Clinical features common to all cases were a history of hypertension, large anteroapical akinetic area with left ventricular dysfunction, acute heart failure culminating in cardiogenic shock, small CK release compared to the estimated area at risk, severe MR, absence of a clear infarct-related coronary artery in 3 out of 4 patients, unresponsiveness to "classical" anti-ischemic therapy aiming at reducing pre and afterload, dramatic resolution of shock using negative inotropic drugs and interrupting vasodilators and mechanical support, and most notably almost complete recovery of left ventricular shape and systolic function at follow-up (Table I).

Some of the latter characteristics suggest mechanisms other than those involved in myocardial infarction (e.g. complete left ventricular function recovery, small CK release, normal coronary arteries) and lead to speculation about an alternative hypothesis.

In case 1 a true myocardial infarction could have been caused by acute left anterior descending artery occlusion, with a compensatory ventricular hyperkinesis causing a dynamic LVOT obstruction, mitral systolic anterior movement and regurgitation. Such a mechanism has previously been proposed[27-29], but the limited CK release and TIMI grade 3 flow at angiography is not in its favor. Alternatively, hypovolemia and acute anemia could have induced the hypercontractile state that, in the presence of anatomical predisposing factors, determined LVOT obstruction; probably afterload increase induced ischemia in the left anterior descending artery territory, aggravating the initial mechanism. The fact that low dose dobutamine reproduced the LVOT obstruction and the kinetic biphasic response supports the hypothesis that ischemia is also due to intraventricular pressure overload. Dobutamine has been known to induce LVOT obstruction during stress echo[11], especially in patients with long standing hypertension with both concentric or mid-septum left ventricular hypertrophy, and can reproduce ischemic symptoms (chest pain and hypotension) and ECG changes also in the absence of significant coronary artery disease[30].

Cases 2 and 3 showed the most dramatic clinical presentation mimicking AMI with shock, even though ECG was not typical in case 2. An ischemic precipitating cause was very unlikely since coronary arteries were normal, without any evidence of spasms or coronary emboli. No clear precipitating cause was evident, but it is our opinion that hypovolemia and hyperkinetic response were the initial factors. The first therapeutic approach contributed to the perpetuation of a vicious circle. The finding of a severe MR in case 2 did not prompt the search for possible mechanisms, but on the contrary the patient was treated with IABP that act-

Table I. Patients' characteristics.

Case	Associated diseases	ECG	Clinical presentation	Coronary angiograms and left ventricle	Rx	
					Before echo	After echo
No. 1	Hypertension Surgery*	↑ ST D_1, D_2, avF, V_2-V_6	HR 90 b/min BP 100/80 mmHg Killip class 2 CK 743/MB 37 U/l	Middle LAD 95% (TIMI 3), Cx and RCA 60% Apical dyskinesis, basal hyperkinesis Sigmoid I-V septum EF 56%, SAM, MR +++	DBT, TNT	β-blockers, ACE-inhibitors
No. 2	Hypertension	↑ ST D_1, avL, V_2 ↓ ST D3, avF	HR 90 b/min BP 100/70 → 50/30 mmHg Shock CK 492/MB 96 U/l	Normal coronary arteries Apical, medium septal and anterior akinesis, basal hyperkinesis EF 35%, SAM, MR +++	IABP, DBT, EPI, TNT	β-blockers
No. 3	Hypertension	LAFB-LVH	HR 100 b/min BP 70/50 mmHg Shock CK 347/MB 62 U/l	Normal coronary arteries Apical akinesis, sigmoid I-V septum LVOT gradient (70 mmHg) EF 35%, SAM, MR +++	DBT, TNT	β-blockers
No. 4	Dialysis** Hypertension	↑ ST V_4-V_6, D1	HR 90 b/min BP 90/50 mmHg Dizziness CK 226/MB 41 U/l	Prox RCA 95% (PTCA), D2 70%, middle LAD and prox Cx 50% Apical dyskinesis, lateral wall hyperkinesis I-V septum 13 mm thick LVOT gradient (180 mmHg) EF 70%	TNT	β-blockers, fluids, soft dialysis

BP = blood pressure; CK = creatine kinase; Cx = circumflex coronary artery; DBT = dobutamine; EF = ejection fraction; EPI = adrenaline; HR = heart rate; IABP = intra-aortic balloon pump; I-V = interventricular; LAD = left anterior descending coronary artery; LVH = left ventricular hypertrophy; LVOT = left ventricular outflow tract; MR = mitral regurgitation; RCA = right coronary artery; SAM = systolic anterior movement; TNT = i.v. nitroglycerin. * hip replacement the day before; ** during hemodialysis.

ed very unfavorably. Tse et al.[31] described a similar case in a female patient, treated with IABP because of an AMI with shock, showing a worsening of clinical conditions until beta-blockers were administered. In our case, IABP was inserted because of the severe MR, and its unfavorable effect is clearly depicted in figure 6. Since case 3 occurred few days after case 2, similar angiographic findings immediately suggested search for the underlying mechanism, and transesophageal echocardiography clearly disclosed the real mechanism. Case 4 recognized a clear mechanism in which dialysis-induced hypotension acted on a hypertrophic left ventricle creating a mid-ventricular obstruction with only ECG signs of ischemia. The presence of a multivessel coronary artery disease did not prevent a reflex hyperkinesis, as recently suggested by Haley et al.[32], especially in the territory of the right coronary artery. Despite the absence of any clinical correlation, PTCA was performed on the right coronary artery.

In conclusion, the link between dynamic LVOT obstruction and ischemic heart disease is difficult to assess, both from an etiopathogenetic point of view and because diagnosis is not easy in the absence of suspicion. Dynamic LVOT obstruction is a reversible mechanism of severe heart failure in the setting of AMI. Its incidence is not negligible: in our Coronary Care Unit these 4 cases accounted for about 15% of all cases of infarction requiring inotropic support. This figure is probably overestimated because of clustering and close presentation of patients to physicians aware of preceding patients, but probably the true incidence is underestimated. However, there are only occasional reports in the literature. Similar cases have recently been reported at the Mayo Clinic[32], although in our series mitral incompetence was the rule, and was clearly related to functional mechanisms, as recently described[33].

It is our opinion that a dynamic rather than an ischemic (coronary thrombus or spasms) cause was the trigger involved in mimicking the classic signs and symptoms of anterior myocardial infarction, although the presence of coronary stenosis (cases 1 and 4) can aggravate it. The absence of a predisposing cause of coronary embolism (e.g. atrial fibrillation) makes this hypothesis very unlikely.

We have seen that hypertension (even in the absence of overt hypertrophy) constitutes a substrate for LVOT obstruction because of left ventricular geometry changes (sigmoid septum) and hyperkinetic response. Ischemic symptoms (chest pain) and ECG changes are more likely due to acute apex pressure overload and expansion rather than coronary occlusion. Drug-induced pre and/or afterload reduction perpetuates the dynamic mechanism. CK release is small and probably originates from subendocardial damage. From the diagnostic point of view, the presence of such a mechanism should be postulated in hypertensive patients with anterior myocardial infarction presenting with severe MR, trivial CK release, and heart failure. The absence of coronary occlusion should immediately raise the suspicion of a dynamic mechanism. An accurate echocar-

diographic examination is essential to disclose LVOT obstruction. Transesophageal approach in patients with a less than optimal acoustic window or who have been intubated is clearly superior to the conventional approach in this setting[19,34-36]. Once the presence of a dynamic LVOT obstruction is confirmed, therapy must be immediately turned towards negative inotropic drugs (i.e. beta-blockers), volume expansion and avoidance of arterial and venular vasodilators and IABP. Alpha receptor agonists (i.e. norepinephrine) are a good alternative, but may lead to coronary vasoconstriction. Clinical course was then benign with an almost complete recovery of left ventricular function, and none of the patients subsequently showed residual LVOT obstruction at rest.

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