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Citation

Cabot, Richard C., Nancy Lee Harris, Jo-Anne O. Shepard, Eric S. Rosenberg, Alice M. Cort, Sally H. Ebeling, Christine C. Peters, et al. 2010. "Case 40-2010." *New England Journal of Medicine* 363 [27] (December 30): 2652–2661. doi:10.1056/nejmcpc1011317.

Published Version

doi:10.1056/NEJMcp1011317

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Case 40-2010: A 68-Year-Old Woman with Chest Pain during an Airplane Flight

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 and R. Neal Smith, M.D., Ph.D.

PRESENTATION OF CASE

From the Cardiology Division, Department of Medicine (M.H.P., K.R.), and the Departments of Radiology (S.D.) and Pathology (R.N.S.), Massachusetts General Hospital; and the Departments of Medicine (M.H.P., K.R.), Radiology (S.D.), and Pathology (R.N.S.), Harvard Medical School — both in Boston.

N Engl J Med 2010;363:2652-61.
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Dr. Shveta Raju (Medicine): A 68-year-old woman with a history of hypertension and hyperlipidemia was admitted to the hospital because of substernal chest pain.

The patient had been well until approximately 24 hours before admission, when substernal chest pain and pressure developed gradually during an international airplane flight from the Middle East to Europe. The pain did not radiate, and it fluctuated in intensity and resolved spontaneously after several hours. Chest pain recurred during the subsequent flight to the United States; the patient fell asleep, and on arrival in Boston, the pain had resolved. Late that evening, approximately 3 hours before admission, substernal chest pain recurred, associated with diaphoresis and weakness. The blood pressure, measured by the patient's daughter, a physician, was 250/140 mm Hg. The patient took amlodipine and atenolol, which she had not taken while traveling. The chest pain improved but recurred 1 hour later. Her daughter took her to the emergency department at another hospital.

On arrival, the patient reported that the pain was moderately severe and not associated with nausea, vomiting, shortness of breath, or palpitations. The blood pressure was 156/77 mm Hg, the pulse 58 beats per minute, the oral temperature 36.1°C, the respiratory rate 16 breaths per minute, and the oxygen saturation 98% while the patient was breathing ambient air. She was lethargic and appeared to be in pain. The jugular veins were not distended, and the remainder of the examination was normal. The complete blood count was normal; other specimens could not be analyzed because of hemolysis. An electrocardiogram (ECG) reportedly showed sinus rhythm at 60 beats per minute, with ST-segment elevation of 2 mm in leads V₂ through V₆, I, and aVL. A chest radiograph showed perihilar fullness with indistinct pulmonary vasculature suggestive of interstitial pulmonary edema. Metoprolol, morphine sulfate, eptifibatid, heparin, and nitroglycerin were administered intravenously, and acetylsalicylic acid was given orally. Less than 1 hour later, the blood pressure was 129/73 mm Hg and the pulse 65 beats per minute. The patient was transferred to this hospital, arriving approximately 3 hours after the onset of the most recent episode of chest pain.

On arrival in the emergency department, the patient reported chest discomfort, which she rated at 8 or 9 on a scale of 1 to 10, with 10 indicating the most severe

pain. Five years earlier, results of a cardiac stress test and cardiac computed tomography (CT) were reportedly normal. She was born in Eastern Europe, lived in the Middle East, and did not speak English. She did not smoke, drink alcohol, or use illicit drugs. Medications at home included atenolol, amlodipine, and atorvastatin. She was allergic to penicillin, atropine, and iodine.

On examination, the blood pressure was 175/85 mm Hg, the pulse 72 beats per minute, and the respiratory rate 18 breaths per minute. The skin was slightly cool. Carotid-artery upstrokes were normal. The jugular veins were moderately distended. The S_1 and S_2 heart sounds were normal; an S_4 , with no S_3 or murmur, was heard, and the remainder of the examination was normal. A chest radiograph revealed findings that were consistent with mild-to-moderate interstitial pulmonary edema and bilateral pleural effusions. An ECG revealed sinus rhythm at 60 beats per minute, a normal axis, and ST-segment elevation of 2 mm in leads V_2 through V_6 , I, and aVL, with Q waves and T-wave inversions in those leads.

Cardiac catheterization was performed 14 minutes after the patient's arrival. Coronary angiography revealed a right-dominant system; a short left main coronary artery with 20% stenosis; proximal 70% stenosis and complete occlusion of the middle portion of the left anterior descending (LAD) coronary artery, with poor collateral flow to the distal LAD artery; and an eccentric lesion in the ostium of second diagonal artery, with 70% stenosis and no thrombus. The right coronary artery was normal. A temporary pacemaker and an intraaortic balloon pump were placed. Thrombectomy of the proximal and middle portions of the LAD artery was attempted, followed by the placement of overlapping bare-metal stents, with no residual occlusion. Balloon angioplasty was performed on the ostial diagonal lesion; the residual stenosis decreased to 30%. Flow defined as grade 2 according to the Thrombolysis in Myocardial Infarction (TIMI) scale (ranging from 0 to 3, with 0 indicating no flow and 3 normal flow) was achieved at the areas of intervention. Medications administered in the catheterization laboratory included clopidogrel, heparin, nitroglycerin, eptifibatide, fentanyl, and vancomycin.

The white-cell count, platelet count, and levels of electrolytes, total and direct bilirubin, total protein, albumin, globulin, phosphorus, and alkaline phosphatase were normal; other laboratory-test

results are shown in Table 1. ECG after the procedure revealed persistent, but decreased, ST-segment elevation in the anterolateral leads. The patient was transferred to the coronary care unit 2 hours after admission. Aspirin, clopidogrel, eptifibatide, metoprolol, acetylcysteine, omeprazole, ondansetron, and acetaminophen were administered. Seven hours after catheterization, laboratory tests were repeated (Table 1).

In the afternoon of the first hospital day, results of arterial blood gas analysis performed while the patient was breathing 2 liters of oxygen by nasal cannula showed a pH of 7.30, with partial pressures of carbon dioxide and oxygen of 33 mm Hg and 127 mm Hg, respectively, and 98% oxygen saturation. Approximately 13 hours after admission, a transthoracic echocardiogram, obtained while the intraaortic balloon pump was providing left ventricular support, showed a left ventricular ejection fraction (LVEF) of 35% (reference range, >50), a normal-size left ventricular cavity, moderate dysfunction involving the septal and anterior segments of the midportion of the left ventricle, and akinesis of the left ventricular apex. There was compensatory hyperkinesis at the base of the left ventricle and narrowing of the left ventricular outflow tract during systole, with a resting systolic left ventricular outflow tract gradient of 31 mm Hg according to Doppler imaging. There was no left ventricular thrombus. A small pericardial effusion was present, with no evidence of tamponade.

On the evening of the first day, chest pain transiently recurred, and an ECG showed increased ST-segment elevation. Repeat coronary angiography 17 hours after admission showed that the stents were patent. ECG after catheterization and 9 hours later showed normal sinus rhythm, with persistent ST-segment elevation.

On the second day, oral metoprolol was added. On the third day, the hematocrit decreased to 24.5%; 2 units of red cells were transfused. The intraaortic balloon pump was removed on the morning of the fourth day. At 2:30 p.m. that day, the blood pressure decreased transiently, with a mean arterial pressure of 58 mm Hg, without chest pain or light-headedness, and no change in the heart rate; the ECG was unchanged. Cultures of the blood and urine were obtained, and the metoprolol dose was decreased. That evening, after eating dinner, the patient reported mild dyspepsia. Shortly thereafter, she stated to her daughter

Table 1. Laboratory Data.*

Variable	Reference Range, Adults†	1st Day, during Cardiac Catheterization	1st Day, 7 Hr after Cardiac Catheterization	2nd Day
Hematocrit (%)	36.0–46.0 (women)	30.4		27.7
Hemoglobin (g/dl)	12.0–16.0 (women)	10.2		9.5
Activated partial-thromboplastin time (sec)	22.1–34.0	>150.0		42.4
Prothrombin time (sec)	10.3–13.2	15.2		13.7
International normalized ratio		1.4		1.2
Troponin T (ng/ml)	0.00–0.09	0.68	14.96	5.83
Creatine kinase MB isoenzymes (ng/ml)	0.0–6.9	26.1	225.4	65.6
Creatine kinase (U/liter)	40–150 (women)		4476	2546
Magnesium (mg/dl)	1.7–2.4		3.5	2.4
Calcium (mg/dl)	8.5–10.5		7.7	7.9
Aspartate aminotransferase (U/liter)	9–32	437		357
Alanine aminotransferase (U/liter)	7–30	72		148
Total cholesterol (mg/dl)	<200, desirable	240		
Low-density lipoprotein (mg/dl)	<130, desirable	169		
High-density lipoprotein (mg/dl)	35–100	39		
Triglycerides (mg/dl)	40–150	158		
Arterial blood				
Fraction of inspired oxygen			Not specified	0.4
pH	7.35–7.45		7.31	7.45
Partial pressure of carbon dioxide (mm Hg)	35–45		39	31
Partial pressure of oxygen (mm Hg)	80–100		167	89
Oxygen saturation (%)			99	97
Base excess (mmol/liter)			6.2, negative	2.5, negative

* To convert the values for magnesium to millimoles per liter, multiply by 0.4114. To convert the values for calcium to millimoles per liter, multiply by 0.250.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

that she did not feel well; the pulse decreased to the 50s, with increased ST-segment elevations, followed quickly by the sudden onset of severe chest pain. Within seconds, the blood pressure could not be obtained, and pulseless electrical activity and electromechanical dissociation developed. Cardiopulmonary resuscitation measures were instituted immediately; the trachea was intubated, and epinephrine, glucose, bicarbonate, insulin, calcium chloride, normal saline, packed red cells, and atropine were administered. Echocardiography revealed a large pericardial effusion. Pericardiocentesis was performed, and 750 to 800 ml of red, turbid fluid was aspirated, with a

hematocrit of 17.5%. Echocardiography showed a decrease in the effusion, but there was no measurable pulse or blood pressure; after 36 minutes, resuscitation efforts were stopped and the patient died, 90 hours after admission.

An autopsy was performed.

DISCUSSION OF MANAGEMENT

Dr. Michael H. Picard: In the case of this 68-year-old woman in whom cardiogenic shock developed several days after acute anterior myocardial infarction with ST-segment elevation, several points are worth discussing: how to handle chest pain

in a passenger on an airplane, how to treat acute myocardial infarction with ST-segment elevation, and the cause of this patient's cardiogenic shock.

IN-FLIGHT MEDICAL EMERGENCIES

Cardiac conditions are among the most frequent medical problems encountered on aircraft and the most common reason for diversion of aircraft.^{1,2} If the patient had reported her symptoms to the staff and a physician had been on board, the differential diagnosis would have included angina pectoris from coronary artery disease, a pulmonary embolus, atypical chest pain with a musculoskeletal origin, an aortic dissection, and other less probable causes. Ideally, if the level of suspicion for an acute coronary syndrome, a pulmonary embolism, or acute aortic dissection had been high, the plane would have landed and the patient would have been transported to a hospital as quickly as possible. For a suspected acute coronary syndrome and normal or high blood pressure, aspirin and sublingual or oral spray nitroglycerin preparations from the in-flight medical kit could have been given. Even if the symptoms resolved, formal medical evaluation on landing would have been recommended.

INITIAL THERAPY FOR ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

Myocardial infarction with ST-segment elevation typically occurs when an occlusive thrombus forms on a ruptured atherosclerotic plaque. The waxing and waning chest pain in this patient probably represented periods of variable blood flow before the thrombus became totally occlusive. The outcome depends on reestablishing coronary blood flow in the affected artery, the time it takes to achieve this blood flow, and whether the artery then remains patent. The goal of initial therapy in ST-segment elevation myocardial infarction is to minimize the time from the onset of symptoms to the initiation of reperfusion therapy. The exact duration of this patient's ST-segment elevation myocardial infarction is unknown. If the patient had had symptoms for less than 12 hours and ST-segment elevation persisted on ECG, reperfusion therapy would typically be recommended in addition to antiplatelet and antithrombin therapy. If there is access to a catheterization laboratory and the artery can be mechanically opened within 12 hours with the use of percutaneous devices, that approach is typically recommended.

Otherwise, treatment with thrombolytic therapy is recommended if it can be administered within the time window — current goals are for percutaneous coronary interventions to be achieved within 90 minutes after presentation at the hospital or for fibrinolytic therapy to be initiated within 30 minutes after arrival.³ Once medical attention was sought, this patient was quickly transferred to our center for intervention. Before the transfer, she received appropriate medications, including beta-blockade, aspirin, heparin, a glycoprotein IIB/IIIa inhibitor, nitroglycerin, and pain control with morphine.

Dr. Kenneth Rosenfield: In the emergency room, this patient had persistent pain, which was consistent with ongoing infarction. Because she was presumed to have salvageable myocardium and was hemodynamically unstable, we accepted her directly to the cardiac-catheterization laboratory for angiography and possible revascularization. The initial injection of the left coronary artery, performed in the right anterior oblique projection (Fig. 1A and Video 1), shows occlusion of the LAD artery about one third of the way into its course. The left circumflex coronary artery and its branches appear to be free of obstruction. The right coronary artery is dominant and not obstructed. There is poor collateral flow to the distal LAD artery.

A temporary pacemaker wire was placed prophylactically. After advancement of a guidewire through the occluded segment of the LAD artery, an uninflated balloon was passed through the lesion and then withdrawn, to reestablish flow and allow us to assess the characteristics of the lesion. There was a critical stenosis in the proximal segment of the vessel, associated with a large thrombus and sluggish flow to the distal LAD artery. We tried to use a suction catheter to extract the thrombus, which is standard treatment for acute myocardial infarction, but we were unable to pass the catheter and therefore proceeded directly to balloon angioplasty. After the initial dilation with a 2-mm balloon, moderate hypotension developed; an intraaortic balloon was placed both for hemodynamic support and to optimize perfusion into the revascularized coronary artery. We placed two overlapping bare-metal stents (each with a diameter of 2.75 mm) in the lesion. A diagonal branch arising within the stented segment was partially compromised at its origin. A guidewire was advanced through the side struts of the



Videos showing coronary angiography and transthoracic echocardiography are available at NEJM.org

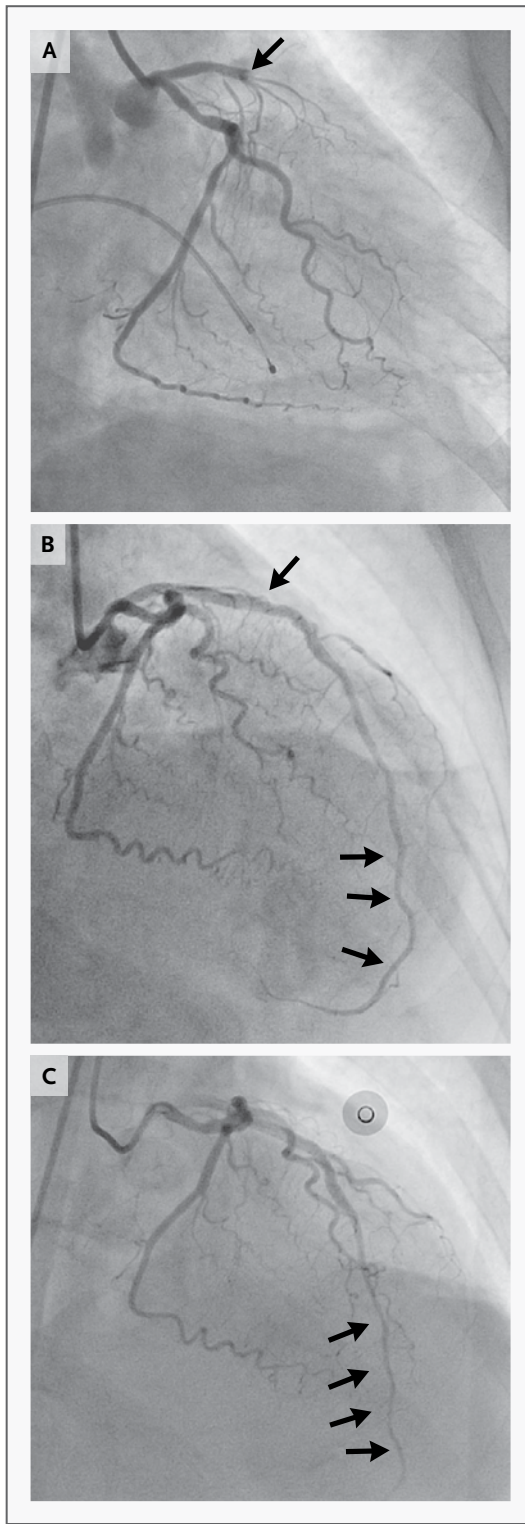


Figure 1. Coronary Angiographic Studies.

A still-frame image from the angiogram obtained on presentation (Panel A), from the caudal right anterior oblique projection, shows the left coronary-artery system. The left anterior descending (LAD) coronary artery is totally occluded in its proximal portion (arrow), whereas the left circumflex coronary artery and its obtuse marginal branches are patent. An image from a coronary angiogram of the left coronary-artery system, obtained from the cranial right anterior oblique projection, shows successful stent placement (Panel B, upper arrow) in the proximal LAD artery. Flow has been reestablished to the LAD artery, and opacification of the distal vessel (lower arrows) is now observed. An image from a coronary angiogram obtained 17 hours after presentation (Panel C), when the patient had recurrent chest pain and new ST-segment elevation on electrocardiography, shows that the LAD artery, seen here in the cranial right anterior oblique projection, remains patent. However, flow to the distal vessel is slow or sluggish, and small branch vessels are under-filled, suggesting that perfusion of the capillary bed in the affected territory (arrows) is poor.

and Video 2) shows excellent patency and antegrade flow in the LAD artery and diagonal branch. The flow to the distal vessel is slightly more sluggish (between TIMI grades 2 and 3) than that to the adjacent circumflex artery, suggesting persistent impairment of perfusion at the microvascular level.

Dr. Subba Digumarthy: A chest radiograph (Fig. 2) was obtained with a portable device while the patient was in the supine position. There are low lung volumes. There is an intraaortic balloon pump terminating 3 cm below the top of the aortic arch. The cardiac silhouette is within normal limits for a portable radiograph. There is bilateral symmetric hilar fullness and indistinctness of pulmonary vasculature. There are bilateral small pleural effusions. These findings are most consistent with interstitial pulmonary edema.

Dr. Picard: The blood samples obtained during cardiac catheterization show elevated cardiac biomarkers, confirming the acute myocardial infarction. It was of interest that levels of the enzymes aspartate aminotransferase and alanine aminotransferase were elevated; since these are noted to rise later than the more cardiac-specific enzymes, this would support the concern that the infarction process had been occurring for a longer period of time than initially thought. At 7 hours after the catheterization, a rise was documented in the levels of the cardiac enzymes, a feature that was consistent with the washout of these en-

stent into the diagonal branch, and we inflated the balloon within the narrowed site to “rescue” the diagonal branch. Final angiography (Fig. 1B

zymes from infarcted myocytes after blood flow was reestablished, possibly with some degree of periprocedural myonecrosis.

TRANSTHORACIC ECHOCARDIOGRAPHY

The echocardiogram obtained on the first hospital day revealed regional dysfunction of the left ventricle in the territory supplied by the LAD artery (Videos 4 and 5). The apex of the left ventricle was dilated, and the LVEF was 35%. Regional left ventricular dysfunction, as seen on this echocardiogram, is the hallmark of myocardial ischemia, infarction, or both. When blood flow is reestablished promptly, a recovery of some or all of the regional function is anticipated; however, this will occur over a period of hours and depends on the duration of the occlusion of the artery. Since the assessment in this patient was performed approximately 12 hours after the intervention, recovery of some additional function could occur; it is not yet possible to predict what the ultimate ventricular regional and global function will be.⁴ However, the extent and degree of dysfunction are quite large, and I would predict that a significant infarct will remain. A small pericardial effusion is also present. Another notable finding is that the myocardial function at the base of the heart was so vigorous that it caused a narrowing of the outflow tract of the left ventricle, with a resulting pressure gradient during ventricular ejection. This dynamic pressure gradient may have been further increased by the reduction in afterload induced by intraaortic balloon pumping.

Dr. Rosenfield: Seventeen hours after admission, because of the patient's recurrent chest pain, we repeated the angiography to confirm that the stents were patent (Fig. 1C and Video 3). The LAD artery remained patent, but flow to the distal portions of the vessel was even more sluggish than after initial revascularization. The myocardial blush was also diminished, indicating poor capillary filling and poor perfusion at the myocyte level. At that time, I concluded that a substantial volume of the tissue that I had revascularized had already completed its infarction and was not salvageable, despite restoration of normal flow. That observation also drove my decision to leave the balloon in place for a longer time, which I hoped would reduce the risk of a left ventricular aneurysm, allow for the administration of agents (e.g., angiotensin-converting-enzyme inhibitors) to reduce afterload, and reduce the risk of a rupture of the left ventricle.

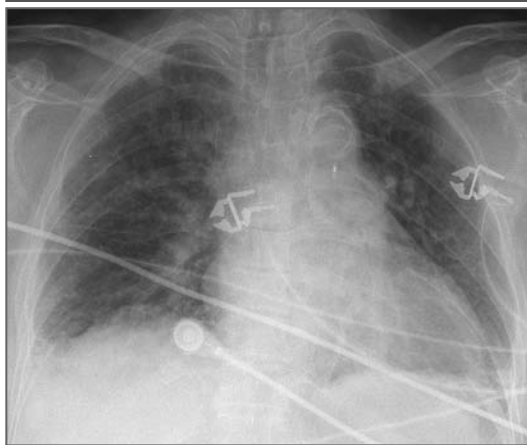


Figure 2. Portable Chest Radiograph.

The pulmonary vasculature is indistinct, and there are bilateral small pleural effusions. An intraaortic balloon pump terminates below the top of the aortic arch.

DIFFERENTIAL DIAGNOSIS

Dr. Picard: Despite optimal management, cardiogenic shock developed on the fourth hospital day. The differential diagnosis of shock could include noncardiac causes such as hypovolemia or sepsis, but the bradycardia, increase in ST-segment elevations on ECG, and recurrence of severe chest pain before the onset of hypotension, with progression to electromechanical dissociation, point us quickly to a cardiac cause (Table 2).

OBSTRUCTION OF THE LEFT VENTRICULAR OUTFLOW TRACT

I have included obstruction of the left ventricular outflow tract in the differential diagnosis, because the transthoracic echocardiogram obtained on day 1 showed changes consistent with a pressure gradient in the left ventricular outflow tract that were due to hyperdynamic contraction of this portion of the left ventricular myocardium.⁵ It would be important to determine whether such obstruction was the cause of the shock, since inotropic agents that might be used to treat other causes of hypotension and shock would exacerbate the obstruction. It is unlikely that left ventricular outflow tract obstruction was the cause of shock in this case. First, usually the compensatory hyperkinesis of unaffected portions of the left ventricular myocardium decreases during the first few days after myocardial infarction. Second, a hyperdynamic state would typically be accompanied by a tachycardia rather than the bradycardia noted in

Table 2. Cardiac Causes of Shock after Myocardial Infarction.

Obstruction of the left ventricular outflow tract
Severe right ventricular dysfunction
Right ventricular myocardial infarction
Pulmonary embolus
Cardiac tamponade from hemorrhagic pericarditis or pericardial hematoma
Recurrent ischemia leading to severe cardiac dysfunction
Mechanical complication
Rupture of the papillary muscle
Myocardial perforation
Pseudoaneurysm
Rupture of the ventricular septum
Rupture of the free wall of the ventricle

this patient. Third, afterload would increase after removal of the intraaortic balloon pump, thus reducing the potential for obstruction. Finally, this patient had received a transfusion, which would also improve left ventricular volume and reduce obstruction.

PULMONARY EMBOLUS

In the distant past, massive pulmonary embolism accounted for a significant number of deaths due to myocardial infarction. Although her long plane flights and prolonged bed rest put this patient at risk for a pulmonary embolus, she had been receiving therapeutic anticoagulation therapy, which should prevent venous thrombus formation. Also, a pulmonary embolus would typically present with tachypnea and tachycardia rather than bradycardia.⁶

CARDIAC TAMPONADE

Pericardial effusions can occur after anterior ST-segment elevation myocardial infarction but typically do not lead to hemodynamic compromise unless they are caused by rupture of the free wall of the ventricle or hemorrhagic pericarditis. The likelihood that this patient had hemorrhagic pericarditis that evolved into cardiac tamponade is low, since classic symptoms of pericarditis did not develop and the ECG changes were localized rather than diffuse and did not evolve in the classic manner for pericarditis. The main reason to consider cardiac tamponade is that with transmural infarctions, there is commonly inflammation of the adjacent pericardium, and since this patient received anticoagulation and antiplatelet

therapy, she would have been at increased risk for hemorrhagic conversion of pericarditis. Dressler's or post-myocardial infarction syndrome could present with a pericardial effusion, but this typically occurs later than the fourth day after the myocardial infarction and rarely would progress to hemodynamic collapse.

RECURRENT MYOCARDIAL ISCHEMIA

Recurrent ischemia either within the territory of the LAD artery or involving another territory could result in a substantial area of additional ventricular dysfunction resulting in hypotension. Of additional concern is the risk of early stent thrombosis. Although the patient was taking medications that guard against coronary stent thrombosis, the recurrence of chest pain and worsening of ECG changes raise the suspicion of recurrent myocardial ischemia.

MECHANICAL COMPLICATIONS AFTER MYOCARDIAL INFARCTION

Of utmost concern in this patient is a mechanical complication after myocardial infarction. One such complication is perforation of a ventricular wall (e.g., rupture of the free wall, the ventricular septum, or a myocardial wall contained by adherent pericardium, resulting in a pseudoaneurysm). Rupture of a papillary muscle, leading to acute severe mitral regurgitation, must also be considered and, in studies of cardiogenic shock, was the most common mechanical complication.⁷ Mechanical complications most often occur in the first week after myocardial infarction. Risk factors include a first myocardial infarction without a history of angina, female sex, advanced age, hypertension, delayed recognition of myocardial infarction, and prolonged physical activity during myocardial infarction. This patient had many of these risk factors.^{8,9}

When there is suspicion of a mechanical complication after myocardial infarction, an echocardiogram obtained immediately, as in this case, can narrow the differential diagnosis and increase the speed to the correct diagnosis and treatment.¹⁰ Most of the conditions in Table 2 have unique features on ultrasonography.

RUPTURE OF THE FREE WALL OF THE LEFT VENTRICLE

Since the echocardiogram shows that a large pericardial effusion is present, we can narrow our dif-

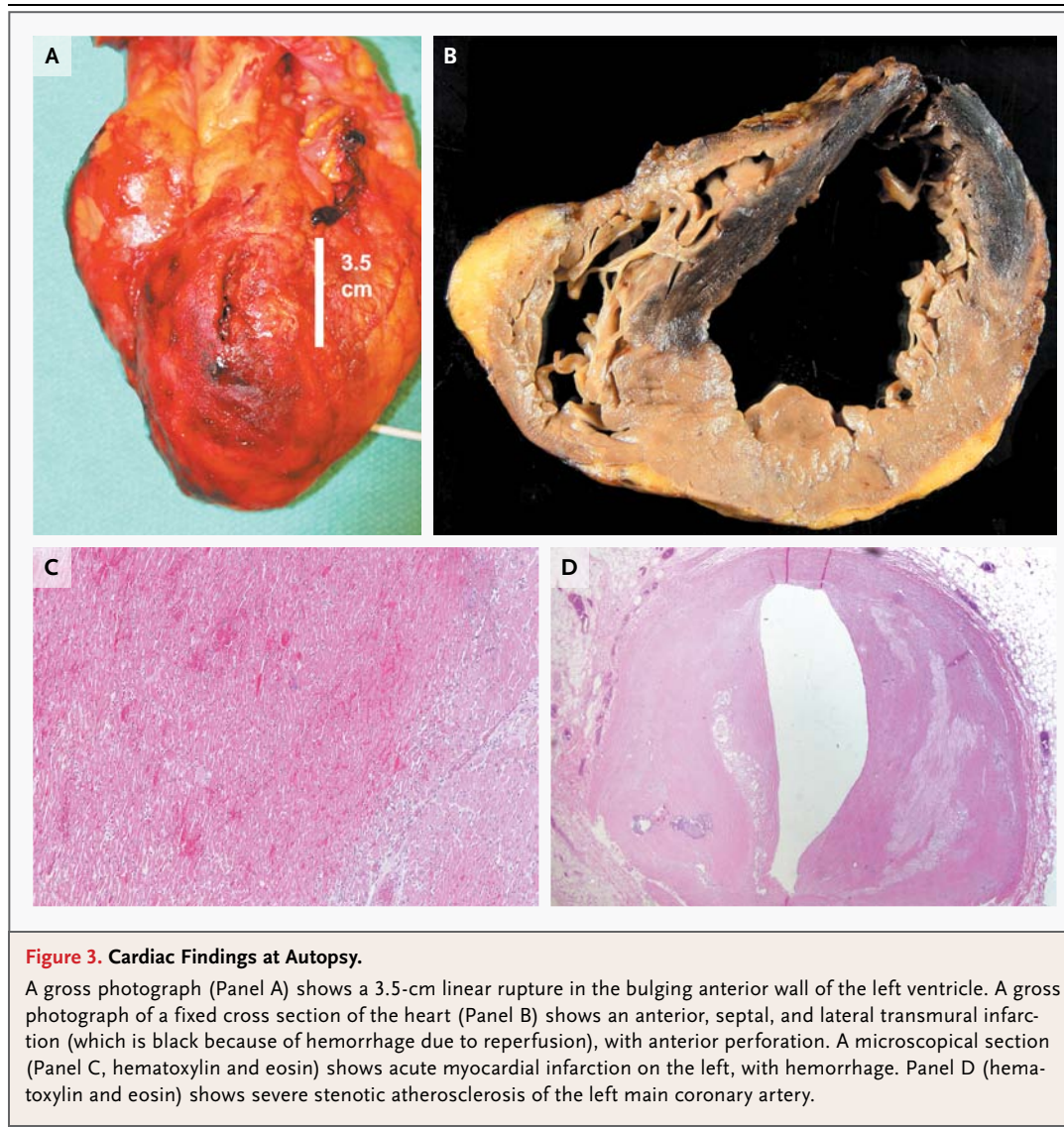


Figure 3. Cardiac Findings at Autopsy.

A gross photograph (Panel A) shows a 3.5-cm linear rupture in the bulging anterior wall of the left ventricle. A gross photograph of a fixed cross section of the heart (Panel B) shows an anterior, septal, and lateral transmural infarction (which is black because of hemorrhage due to reperfusion), with anterior perforation. A microscopical section (Panel C, hematoxylin and eosin) shows acute myocardial infarction on the left, with hemorrhage. Panel D (hematoxylin and eosin) shows severe stenotic atherosclerosis of the left main coronary artery.

ferential diagnosis to rupture of the free wall and other causes of cardiac tamponade. Rupture of the free wall of the left ventricle occurs in 1 to 4% of cases of acute myocardial infarction. It commonly presents as acute or recurrent chest pain followed by electromechanical dissociation, as in this case.^{11,12} Examination may reveal jugular venous distention, hypotension, pulsus paradoxus, and reduced intensity of heart sounds. When blood first enters the pericardial space, there may be a brief period of increased vagal tone, with bradycardia, diaphoresis, and nausea; this patient reported dyspepsia before the onset of pain and hypotension. Rupture of the myocardium typically occurs at the terminal portion of a coronary-artery dis-

tribution, so in this case, we would predict that it would have occurred in the anterolateral wall of the left ventricle. The best treatment is immediate surgery, although the prognosis remains poor, since it is challenging to get such critically ill patients to the operating room in time. Pericardiocentesis rarely leads to stabilization of a patient's condition and, if performed, will show that the fluid is blood, as was shown in this case. If the pericardiocentesis relieves the tamponade, the relief will usually be transient, since the hypotension and tamponade will often resume as blood continues to enter the pericardial space from the left ventricle. It is most likely that this patient died from an acute rupture of the free wall of the left

ventricle 4 days after acute anterior ST-segment elevation myocardial infarction.

Dr. Nancy Lee Harris (Pathology): Dr. Rosenfield, would you give us your clinical impressions at the time?

Dr. Rosenfield: This event was very dramatic. Despite our best efforts, resuscitation was unsuccessful. For the reasons articulated by Dr. Picard, I also favored the diagnosis of acute rupture of the free wall of the myocardium.

CLINICAL DIAGNOSIS

Acute rupture of the free wall of the left ventricle.

DR. MICHAEL H. PICARD'S DIAGNOSIS

Acute rupture of the free wall of the left ventricle 4 days after acute anterior ST-segment elevation myocardial infarction.

PATHOLOGICAL DISCUSSION

Dr. R. Neal Smith: At autopsy, the heart weighed 310 g with approximately 300 ml of pericardial blood. A 3.4-cm transmural rupture (Fig. 3A) is present in the anterolateral surface of the left ventricle and is also identified on cross section (Fig. 3B) in association with a large transmural anterolateral and anteroseptal acute myocardial infarction (Fig. 3B). Histologically (Fig. 3C), an acute anterior, septal, and lateral transmural infarct is present with hemorrhage due to reperfusion after the thrombectomy. The right coronary artery was dominant, and there was near-occlusive complex atherosclerosis of the left main, LAD, and circumflex arteries (Fig. 3D). The pathological diagnoses are severe coronary artery disease of the left main, LAD, and circumflex arteries, with mid-to-proximal occlusion of the LAD artery, after stent placement; acute transmural anterior, septal, and lateral myocardial infarction, 4 days old; and anterior-wall rupture, hemopericardium, and

tamponade. These findings occurred in a patient with risk factors for rupture that included older age, female sex, no previous coronary events, and decreased collateral perfusion.^{13,14}

Dr. Harris: Dr. Rosenfield, if you had been sure that this patient's myocardial infarction had been going on for about 24 hours before she came to the emergency department, would you have performed percutaneous coronary intervention?

Dr. Rosenfield: If I had been certain that the patient's tissue had completed its infarction, then I probably would not have performed immediate revascularization, not because of increased risk but because of a lack of documented benefit.¹⁵ However, in many patients, the status of the myocardium is not certain. This patient's history was of waxing and waning pain, culminating in recurrence of severe pain after her arrival at home. Since her pain largely resolved after stenting, there was most likely salvageable myocardium. If there is even a moderate amount of salvageable myocardium, restoration of normal perfusion improves both the short-term and long-term prognoses.

ANATOMICAL DIAGNOSIS

Myocardial infarction and rupture of the free wall of the left ventricle.

This case was presented at the Medical Case Conference, June 26, 2009.

Dr. Rosenfield reports being a member of the board of VIVA Physicians (a not-for-profit educational organization with support from commercial and private donors; www.vivapvd.com); receiving consulting fees from Abbott Vascular, Boston Scientific, Boston Biomedical, Baxter Healthcare, Harvard Clinical Research Institute, Micell Technologies, and Complete Conference Management; receiving royalties from Angioguard; and holding stock in Micell Technologies, Icon Medical Corp., Contego, Lumen Biomedical, and Medical Simulation Corp. Dr. Rosenfield also reports that Massachusetts General Hospital has received grant support from Abbott Vascular, Boston Scientific, Baxter Healthcare, Cordis, IDEV Technologies, Medtronic/Invatec, Bard, Lutonix, and Atrium on his behalf. No other potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Dr. Rory Weiner for assistance in organizing the conference, preparing the case history, and providing the echocardiography images.

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