

Case 2 – Sudden Death after Coronary Artery Bypass Surgery in a 49-year-old Female, Diabetic, Dyslipidemic, Hypertensive, Obese Patient

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The patient, a 49-year-old woman, was born in São João da Boa Vista (SP) and lived in Guarulhos (SP), died of cardiac sudden death during rehospitalization for treatment of wound infection after coronary artery bypass surgery.

The patient was asymptomatic until May 22, 2009, when she started to have intense chest pain that radiated to the left arm and was hospitalized with the diagnosis of pain that irradiated acute myocardial infarction. She had diabetes mellitus, hypercholesterolemia, hypertriglyceridemia and arterial hypertension. Her father had died at age 55 due to cerebrovascular accident and her mother was alive at 71 years with no cardiovascular disease.

Ventricular function and coronary anatomy assessment was performed on infarction admission, which took place at another hospital.

Echocardiography (25 May 2009) disclosed aortic diameters of 32 mm, 43 mm left atrium, left ventricle during diastole of 57 mm; left ventricular ejection fraction of 47%, interventricular septum thickness of 11 mm and 10-mm posterior wall. Inferolaterodorsal hypokinesia was observed.

Coronary angiography (May 28, 2009) disclosed a 70% lesion in the anterior descending branch in its middle third and occlusion of the circumflex branch before the posterior ventricular branch. There were no obstructive lesions in the right coronary artery and collateral circulation was observed from the right coronary artery to the circumflex branch; the ventriculography showed lateral akinesia.

After admission, the patient developed dyspnea crises and “wheezing”, as well as angina on moderate exertion.

Keywords

Death, Sudden, Cardiac; Myocardial Revascularization; Hypertension; Obesity; Diabetes Mellitus.

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Physical examination at her first visit to InCor (July 1, 2009) showed weight of 113 kg, height 166 cm, body mass index 41 kg/m², heart rate of 80 bpm, blood pressure 140 x 90 mm Hg. Lung assessment was normal and cardiac auscultation showed a +/4+ systolic murmur in the mitral valve. No abnormalities were found on examination of the abdomen, extremities and neck.

Laboratory assessment (29 July 2009) showed: hemoglobin = 12.7 g/dL, hematocrit = 26.9%, leukocytes = 7150/mm³ and platelets = 348,000/mm³; blood glucose = 186 mg/dL, glycated hemoglobin = 8.8%; urea = 35 mg/dL, creatinine = 0.7 mg/dL, total cholesterol = 109 mg/dL, HDL-C = 43 mg/dL, LDL-C = 38 mg/dL, triglycerides = 141 mg/dL, TSH = 4.74 μU / L and free T4 = 0.86 ng/dL.

The ECG (October 13, 2010) showed sinus rhythm, 57 bpm, PR = 139 MS, DQRS = 79 MS, QT = 392 MS, SÂQRS = +10° and no pathological Q waves or alterations in ventricular rate regularization (Figure 1).

The following were prescribed at the first visit: 10 mg of simvastatin, 100 mg of metoprolol, 100 mg of acetylsalicylic acid, 50 mg of losartan, 20 mg of propatylnitrate, 50 mg of losartan, 12.5 mg of hydrochlorothiazide and 250 mg of chlorpropamide daily.

The interventional cardiologist did not consider the patient as an ideal case for angioplasty and CABG was indicated.

Coronary artery bypass graft surgery (October 14, 2009) consisted of performing terminolateral bypass graft from the left internal mammary artery to the left anterior descending artery, as well as saphenous vein bridge to left posterior ventricular artery. The hospitalization was uneventful, and the patient was discharged on day 7 postoperatively (October 21, 2009) with prescription medications such as those used before surgery, except for simvastatin, which was replaced by 10 mg of rosuvastatin and addition of metformin 500 mg daily.

About two weeks after the surgery, the patient complained of sternum pain and had purulent discharge in the lower third of the sternal region. She had no fever. Cultures were collected from this material, and ciprofloxacin 1 g daily was prescribed (October 27, 2009). Cultures were positive for *Pseudomonas aeruginosa* sensitive to ciprofloxacin and oxacillin-resistant *staphylococcus coagulase-negative*.

Laboratory assessment (October 28, 2010) showed: hemoglobin = 9.8 g/dL, hematocrit = 28.4 g/dL, leukocytes = 9.640/mm³ (1% band cells, 76% neutrophils, 11% lymphocytes and 11% monocytes), platelets = 394.000 /mm³, sodium = 139 mEq/L, potassium = 4.4 mEq/L, urea = 42 mg/dL, creatinine = 1.1 mg/dL, glycemia = 155 mg/dL, glycated hemoglobin = 8.5% and uric acid = 3.8 mg/dL.

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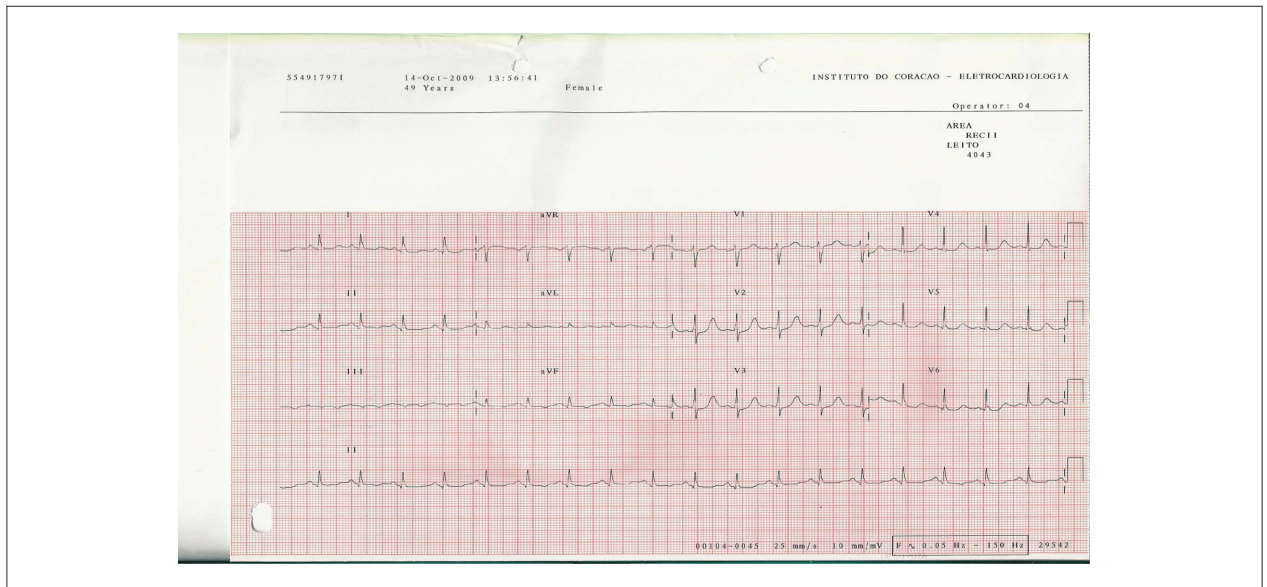


Figure 1 – ECG

Ciprofloxacin was prescribed, but there was no improvement in the secretion and the patient was hospitalized for infection control (November 4, 2009), when teicoplanin was added, at a dose of 800 mg daily.

Physical examination (November 4, 2009) showed heart rate of 68 bp, blood pressure of 130x90 mm Hg, presence of pus in the lower sternal region and mild hyperemia in the graft region.

Laboratory assessment (November 4, 2009) showed hemoglobin = 10.2 g/dL, hematocrit = 32%, leukocytes = 9.800/mm³ (72% neutrophils, 1% eosinophils, 16% lymphocytes and 11% monocytes), platelets = 447.000/mm³, erythrocyte sedimentation rate = 60 mm, CRP = 20.1 mg/L, glucose = 156 mg/dL, urea = 33 mg/dL, creatinine = 1.37 mg/dL (GFR = 43 mL/1.73 m²/min), sodium = 137 mg/dL, potassium = 4.1 mEq/L.

Chest CT scan (November 4, 2009) showed sternotomy with good alignment of bone fragments and discrete blurring of presternal fat planes. Mediastinal vascular structures were of normal caliber and had regular outlines. The trachea and main bronchi were permeable and of normal caliber. There was no lymphadenopathy, but there was cardiomegaly with a small pericardial effusion. Presence of a mild bilateral centrilobular emphysema and nonspecific irregular opacity in the upper segment of the left lower lobe was observed, associated with adjacent pleural thickening. Gallstones and blurring of pararenal fat to the right were also observed. The right kidney showed decreased dimensions.

On the morning after hospital admission she was found lying on the bathroom floor, conscious, with motor deficit, disorientation and, according to the patient, without headache. She had no recollection of how she had fallen. Blood pressure was 98 x 68 mmHg; she was taken to bed and given 0.9% saline and dopamine and 40 min later, she had an episode of pre-syncope with a heart rate of 139 bpm

and blood pressure of 107 x 71 mmHg and O₂ saturation was 94% with an oxygen mask and a few minutes later she had a cardiac arrest unresponsive to resuscitation and died (6:45 a.m. on November 5, 2009).

Clinical aspects

The patient was a 49-year-old woman with myocardial infarction and left ventricular dysfunction submitted to coronary artery bypass graft surgery, who was discharged on the 7th postoperative day. A week later she showed surgical wound infection, which was treated with antibiotics. The patient was later hospitalized with a diagnosis of mediastinitis.

On the morning of the following day, she had a syncope episode in the bathroom; she was conscious, had no motor deficit, was disoriented and had no headache. She did not remember how she had fallen. She had hypotension and tachycardia. Subsequently, she underwent cardiac arrest and died.

This is a case of syncope caused by low cardiac output in a woman with at least five risk factors for pulmonary embolism: age older than 40 years, postoperative period of major surgery, diabetes, obesity and infection^{1,2}. The clinical suspicion of acute pulmonary thromboembolism (PTE) is based on the identification of one or more risk factors and a compatible clinical picture. There is no specific or pathognomonic clinical picture of acute PTE. In most patients with clinically suspected pulmonary embolism, that will not be confirmed and the final diagnosis may point to an alternative condition.

One should always consider the diagnosis of acute PTE in the presence of one of the following clinical scenarios: syncope in the postoperative period in the presence of risk factors; acute chest symptoms in the presence of acute DVT; critically-ill patients or those with trauma; patients with sudden and inexplicable tachyarrhythmias in the presence

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of risk factors; chronic arrhythmia and those presenting with pleuritic pain and sudden hemoptysis, unexplained heart failure decompensation or chronic lung disease, and cardiac arrest²⁻⁶.

In 2007, a study was carried out in Incor-USP, where 406 autopsies were performed, of which 34.1% were diagnosed with pulmonary embolism as the cause of death. The data also showed a divergence in the diagnosis of PTE of approximately 63% between clinical diagnosis and autopsy. There are several hypotheses to justify this discrepancy, as the PTE can manifest as an oligosymptomatic clinical picture and not be recognized, or be so catastrophic as to be mistaken for primary cardiac disease⁷.

Another possible diagnosis is the postcardiotomy syndrome. The patient had undergone surgery two weeks before, referred chest pain in the sternum region with purulent discharge, had increased erythrocyte sedimentation rate and a small pericardial effusion demonstrated by the chest CT.

The postcardiotomy syndrome is a common postoperative complication after cardiac surgery. The etiology points to an autoimmune component related to anti-heart antibodies. The syndrome incidence ranges from 10 to 30% of cases and a decrease in its incidence is observed with increasing age⁸. It occurs between the first and third week after surgery. Patients may present with fever, pleuritic pain in the anterior chest region, pleural or pericardial effusion, pericardial friction rub, increased pain at the surgical incision site, leukocytosis and ESR > 40, as in the case study.

The most important consequences of this syndrome include cardiac tamponade, pericardial constriction and obstruction of grafts and one of these complications may have been the cause of the patient's death.

Sudden cardiac death (SCD) can also be a diagnosis for this case. The presence of ventricular dysfunction in patients with ischemic cardiomyopathy is a potent predictor of SCD. A transient imbalance between supply and demand, either by occlusion or

spasm of the graft postoperatively, may have generated a system of reentry, leading to lethal ventricular arrhythmia.

The long QT syndrome is among the diagnostic options for the patient. When the electrocardiogram was analyzed, we calculated a corrected QT of 550 ms by Bazett's formula. Ciprofloxacin, the antibiotic used to treat patients with mediastinitis may have contributed to the long QT and induced malignant arrhythmia, culminating in syncope and cardiorespiratory arrest (**Dr. George Barreto Miranda**).

Diagnostic hypothesis: acute pulmonary thromboembolism. (**Dr. George Barreto Miranda**)

Necropsy

The heart weighed 540 g and showed left ventricular concentric hypertrophy and myocardial scarring on the diaphragmatic wall (Figure 2). There was coronary atherosclerosis with critical obstructive lesions (> 70%) in the first third of the anterior interventricular branch and distal third of the circumflex branch of the left coronary artery. There was also a recanalized old thrombosis in the distal third of the circumflex branch. The saphenous graft to the posterior interventricular branch showed recent thrombosis that was completely occlusive (Figure 3). The left internal thoracic artery graft (mammery) to the anterior interventricular branch was patent throughout its length. There were no histological signs of recent myocardial ischemia.

The cause of death was massive bilateral pulmonary thromboembolism, with recent thrombus completely occluding the right and left pulmonary arteries at the hilum (Figure 4). Histologically, there were no signs of organization of these thromboemboli.

Other necropsy findings of note were the presence of a small amount of greenish secretion in the surgical wound borders and moderate amount of pus in the facial sinuses, characterizing frontal sinusitis. Histological examination of the surgical wound borders, including the sternum, showed fibrin and leukocyte exudate and necrotic cell debris and no bacteria or fungi (Figure



Figure 2 – Short-axis cut in the ventricular mass, showing infarction scarring on the diaphragmatic surface (arrow).

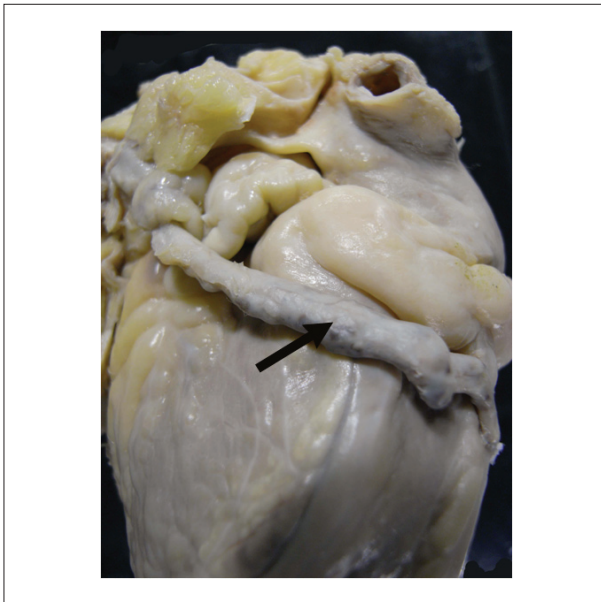


Figure 3 – Left margin of the heart showing the graft course to the posterior interventricular coronary branch. Note the engorged vein (arrow), showing recent thrombosis.

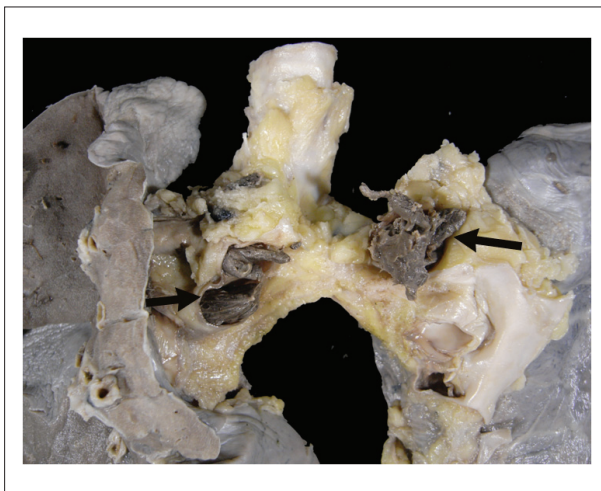


Figure 4 – Frontal view of lung showing hilar branches of the pulmonary arteries completely occluded by recent thrombi (arrows).

5). The study of the spleen showed acute splenitis. The place of origin of the thromboemboli was not determined (**Dr. Vera Demarchi Aiello**).

Anatomopathological diagnoses: ischemic heart disease with myocardial scarring on the diaphragmatic wall; operated coronary atherosclerosis with thrombosis of the saphenous graft to the posterior interventricular branch; purulent frontal sinusitis; acute splenitis (**Dr. Vera Demarchi Aiello**).

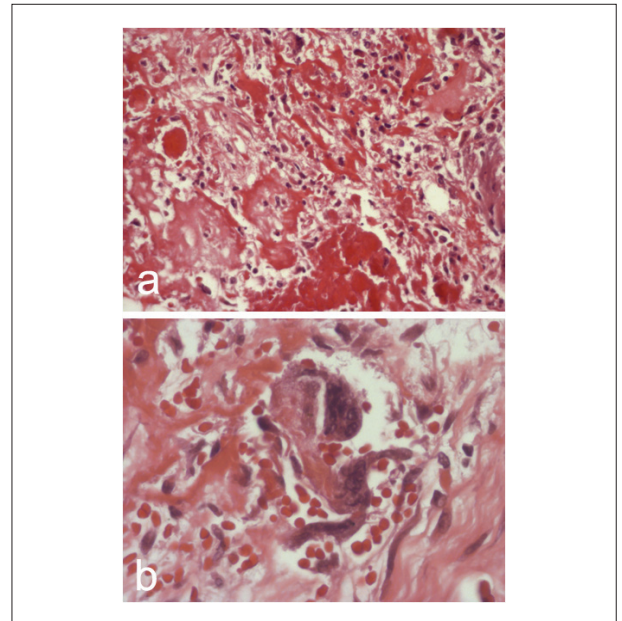


Figure 5 – Photomicrographs of the surgical wound showing in a) necrotic cell debris and mixed inflammatory infiltrate and in b) foreign body-type histiocytic cells, common in surgical suture areas. Hematoxylin-eosin stain, original magnification, 20X and 40X respectively.

Cause of death: recent massive bilateral pulmonary thromboembolism (**Dr. Vera Demarchi Aiello**).

Comments

The patient had been treated by CABG for advanced coronary atherosclerosis, but died from sudden death in the late postoperative period due to massive pulmonary thromboembolism. Several factors can be considered as predisposing to venous thrombosis and thromboembolism postoperatively and among them, immobilization in bed, systemic conditions such as inflammation, neoplastic diseases etc. In an American study that included nearly 200.000⁹ hospitalizations for cardiovascular surgery or invasive procedure it was observed that the incidence of venous thromboembolism varied according to the procedure performed, being 1.2% in those with abdominal aortic aneurysm, 1.1% in patients submitted to limb amputation and 0.54% in those undergoing CABG. The odds ratio for the occurrence of venous thromboembolism was 3.9 in abdominal aortic aneurysm repairs and 1.9 in myocardial revascularizations, when compared to carotid endarterectomy procedure and 1.14 for the female gender¹⁰.

The patient discussed here was diabetic, had purulent sinusitis and suspected wound infection, and, moreover, was overweight. These conditions undoubtedly favored the development of thromboembolism. There was a systemic prothrombotic state, considering the presence of bypass graft thrombosis (**Dr. Vera Demarchi Aiello**).

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