Anatomicoclinical Correlation

Case 4/2005 – Fatal Myocardial Infarction in a 88-year-old woman (Instituto do Coração do Hospital das Clínicas - FMUSP, São Paulo, Brasil)

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An 88-year-old women sought medical care (12/29/03) complaining about a 4-hour duration epigastric pain, followed by vomiting.

The patient had knowledge of her longstanding hypertension, having undergone a cerebrovascular accident two years earlier, without motive sequels. Three months before, dyspnea due to moderate exercise and lower limb edema took place. She denied syncope.

Physical assessment (10/03/03) showed 68-bpm heart rate and blood pressure of 180/80 mmHg. Lung examination did not show any problems, as well as abdomen exam. However, heart examination exhibited normophonetic sounds and mild systolic murmur in the mitral area. A ++/4 edema was identified in the lower limbs.

The patient made daily use of losartan 50 mg, hydrochlorothiazide 50 mg, and acetylsalicylic acid 100 mg.

EKG (09/30/03) displayed atrioventricular dissociation with junctional rhythm, 50-bpm heart rate, 80 msec QRS, with front plan low voltage (figure 1).

Hemoglobin 15 g/dl, hematocrit 45%, potassium of 5.8 mEq/l (hemolized blood) and 138 mEq/l sodium were detected at laboratorial examinations.

Sinus node disease diagnosis was achieved, followed by laboratorial assessment, including ambulatory electrocardiographic monitoring.

Holter 24-hour ambulatory electrocardiographic monitoring (11/18/03) showed prevailing sinus rhythm, 3 ventricular extra-systoles under ventricular tachycardia and a single ventricular extra-systole. Frequent atrial extra-systoles (645/h) took place, from which 570 were isolated, 36 matched, and a 3-heartbeat atrial tachycardia episode. Several under care 4.3-sec sinus pauses, between 8 pm and 9 pm, were detected (figures 2 and 3).

Evolution demonstrated (12/29/03) 4-hour epigastric pain followed by vomiting.

Physical examination (12/19/03) revealed heart rate of 80 bpm, 180/100 mmHg blood pressure, crepitant rales at both hemithorax bases, with no positive data in the remaining of the test.

The EKG (12/29/03) displayed sinus rhythm, 72-bpm heart rate, electrically negative anteroseptal area, an elevated ST segment from V_1 to V_3, and depressed in II, III, aVF, V_5, and V_6 derivations. Changes remained after use of sublingual-administrated 5mg of isosorbide dinitrate (figure 4).

Laboratorial tests showed 1 mg/dl of creatinine, 43 mg/dl of urea, 139 mEq/l of sodium, 5.8 mEq/l of potassium, 49.4 ng/ml of CKMB, and 7.8 ng/ml of troponin.

Myocardial infarction diagnosis was performed and 200mg per oral acetylsalicylic acid and endovenous nitroglycerin were administrated. Blood pressure was controlled and the patient sent to cinecoronariography for mechanical coronary reperfusion through angioplasty.

Cineangiography, 60 minutes after the patient had arrived at the hospital, did not display coronary obstructions. Despite dyskinetic area in left ventricular anterolateral and apical walls, the patient showed asystole cardiopulmonary arrest, without response to resuscitation procedures, and died (12/30/03).

Examination Explanation - graphic methods and imaging: At September 30, 2003 EKG (figure 1) junctional rhythm, 50-heartbeat-per-minute heart rate, SAQRS axle at +30 degrees, 80-msec QRS interval, and 400-msec corrected QT interval were displayed. QRS complexes showed front plan low voltage and ventricular repolarization changes were detected.

Electrocardiogram on 12/29/03 (figure 4) detected sinus rhythm, the presence of supraventricular extra-systole, heart rate of approximately 60 bpm, 140-msec PR interval, and electric heart axle at approximately 0 degree. QRS interval lasted 80 msec. A 3-mm ST segment elevation was found in V_1, V_2, V_3 derivations, and an 1-mm in DI and aVL. Depressed lower wall matches "mirror" imaging. QRS complex amplitude alternation was also detected in DII rhythm record.

(Dr. Paulo Jorge Moffa)

Dynamic Electrocardiogram - Twenty-four-hour EKG recording confirmed sinus node dysfunction diagnosis by showing bradycardia sinus rhythm alternating with atrial ectopic episodes and bradycardia stress periods and up to 4.3 sec pauses, especially between midday and 1pm, 2.20 pm and 3.30 pm, and 8.45 pm and 9.05 pm. ST segment did not show any significant changes (figures 2 and 3).

(Dr. Cesar José Grupi)

Coronary Angiography - Right dominance-type coronary circulation was detected. Right and left coronary arteries and their branches had smooth and obstructive lesion-free walls. Developed anterior descending artery going through anterior interventricular groove, involving apex and extending to posterior interventricular groove.

Left ventriculography displays normal dimension chamber, sho-

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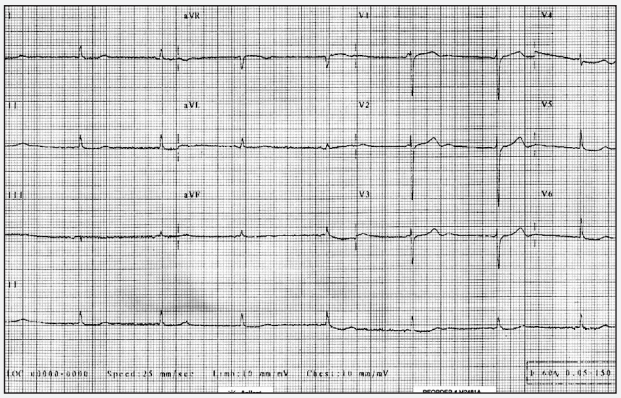

Figure 1 - Electrocardiogram at ambulatory follow-up.

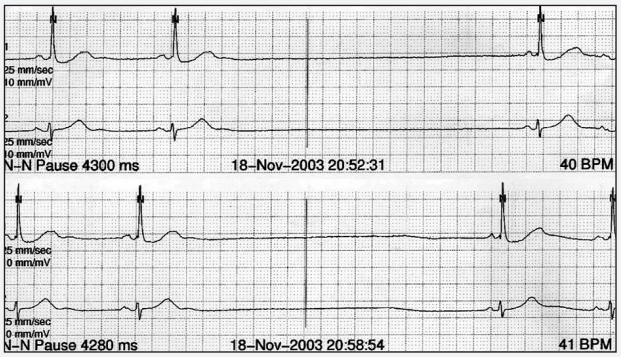

Figure 2 - Ambulatory electrocardiographic monitoring.

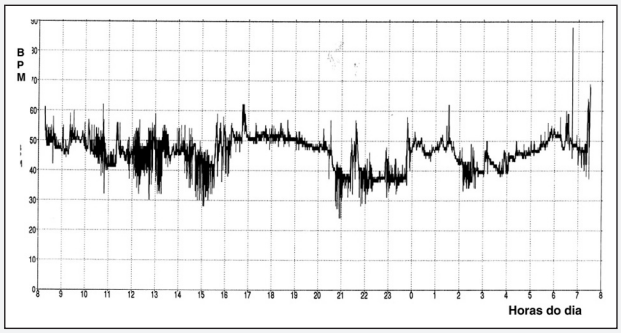

Figure 3 - Ambulatory electrocardiographic monitoring.

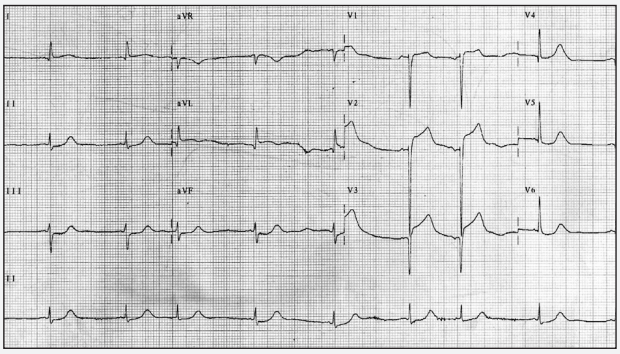

Figure 4 - Electrocardiogram at last admission.

wing anterolateral wall-restricted dyskinetic area, corresponding to an often irrigated area by left coronary artery diagonal branch, with normal contraction of other muscle walls, including at apex. Mild mitral regurgitation is observed.

(Dr. Eulógio Emílio Martinez Filho)

Clinical Features

The case refers to an 88-year-old woman with longstanding systemic hypertension history, cerebrovascular accident two years before, and current sinus node disease diagnosis. Admitted at emergency due to clinical, electrocardiographic and laboratorial features of Killip II anteroseptal wall myocardial infarction with normal coronary arteries at coronary angiography, she evolved to asystole and death on the first post-infarction day.

Normal coronary artery myocardial infarction has been known for more than 60 years, even before coronary arteriography. As long as cardiac catheterization has become a diagnostic/therapeutic instrument in acute coronary syndromes, part of patients could clearly suffer acute myocardial infarction with normal coronary arteries.

Normal coronary artery myocardial infarction frequency appears increased among the female sex and non-Caucasian individuals. GUSTO 1 study demonstrated that 10.2% of women and 6.8% of men with ST elevating myocardial infarction had normal or non-obstructive lesion coronary arteries[1].

Vasospasm, myocardial bridging, acquired or congenital coagulation disorders, toxic agents, embolization, conjunctive tissue diseases, infiltrative diseases, disproportion status between oxygen offer and consumption[2], were among assumptions suggested to explain such entity.

In an etiology-concerned 91 myocardial infarction and normal coronary artery patient study, Da Costa et al.[2] found coronary spasm in 15.5% of patients, coagulation or secondary congenital disorders to contraceptive use in 12.8%, collagen disease in 2.2%, and embolization in 2.2% of patients. Two-thirds of them remained without well-defined cause, even after extensive investigation.

The object individual fits well in the profile of patients with normal coronary artery, ST elevating myocardial infarction patients who show low frequency diabetes mellitus, dyslipidemia, family history of coronary artery disease and obesity when compared to those with significant obstructions[2].

Normal coronary arteries found in cardiac catheterization does not exclude thee presence of atherosclerosis, as those patients may show decreased coronary flow reserve, coronary endothelial dysfunction, and recognizable plaque through endovascular ultrasound imaging. Such findings indicate initial atherosclerosis and correlate with a higher event rate[3].

Vasospasm hypothesis is less probable in the patient due to unchanged clinical features after sublingual nitrate administration.

Many mentioned normal coronary artery myocardial infarction etiologies are little compatible with history, clinical and laboratorial features provided. Neither showed by myocardial bridging. In the patient's age range there is the remote probability of myocardial infarction with elevated ST as first collagen disease manifestation. Furthermore, there are no evidences of drug use causing myocardial infarction.

Coagulation disorder shall be taken into consideration due to patient's cerebrovascular accident history.

Senile amyloidosis represents diagnostic assumption due to our patient be aged, with prior heart failure suggestive complaints, low voltage at EKG, 24-hour Holter suggesting conductive system disease and normal coronary artery acute myocardial infarction.

On the other hand, both coronary artery disease prevalence

and severity increase with age in both genders. Necropsy studies have demonstrated that more than half of over 60-year-old population have coronary artery disease. At 80, 20% to 30% of patients show symptoms, often ischemic equivalent, as dyspnea during exercise, which is present in this case[4].

Embolization hypothesis from cardiac catheterization undetected atherosclerotic with spontaneous recanalization deserves special importance in such context.

Except for the fact that the patient could have shown infiltrative disease, sinus node disease diagnosis does not appear to be related to myocardial infarction, as conductive tissue degeneration is responsible for nearly half of sinus node disease cases in that age range[5]. Neither is atherosclerosis hypothesis reinforced by sinus node disease presence, as ischemia still has had controversial role in disease's etiology.

Important to mention is that among documented coronary failure patients there are accounts of angina functional class worsening unleashed by bradycardia secondary to sinus node disease, as well as worsening of dyspnea during exercise. The most frequent cause of sinus node disease among elderly individuals is related to cell loss and collagen infiltration in sinus node area and the whole atrium. Such changes are greater in sinus node area, with destruction of up to 90% of cells at 75 years of age[5].

Sinus node diagnosis was carried out during ambulatory assessment of the patient based on EKG at rest showing junctional escape rhythm and Holter with presence of several sinus pauses higher than 3 seconds, alternated with tachyarrhythmia episodes.

Such disease is indeed defined in accordance with electrocardiographic criteria, since clinical signs and symptoms may not be present. Dizziness, pre-syncope and syncope are causes that usually make the patient see the physician. However, symptoms as dyspnea during exercise, angina worsening or palpitations can be also present[5].

Sinus node disease comprises any node dysfunction and unsuitable sinus bradycardia characterized by heart rate lower than 60 bpm, which does not properly increase with exercise. Sinus pause longer than 3 seconds, sinus-atrial block, and brady-tachyarrhythmia defined by sinus or junctional bradycardia, alternating with atrial tachycardia or paroxystic atrial fibrillation, are also included. Electrocardiographic expressions may take place either with or without symptoms.

Idiopathic degenerative disease is the most usual cause. Basic conductive system diseases as systemic hypertension, infiltrative disease (amyloidosis), collagen disease, and inflammatory processes can also be possible causes[5]. Effects from sinus node function depressing drugs, such as anti-arrhythmic, beta-blockers, lithium, and amitriptyline, besides endocrine disorders as hypothyroidism, must also be regarded as external sinus node disease causes.

For being an elderly hypertensive woman without sinus node depressive drug use history, intrinsic causes, as degenerative diseases, whose incidence increases with the age, is the most probable cause. There was no account of documented arrhythmia-related symptoms as the patient denied syncope. There was also no reference in Holter report concerning symptoms caused by featured arrhythmias, not even due to sinus pauses under care. As the treatment is symptom-guided, there was no indication of pacemaker placement in this case. Pacemaker implantation, medications, or a combination of them, are therapeutic options for sinus node disease.

Pacemaker is indicated in presence of documented sinus node disease related to symptomatic bradycardia or symptomatic chronotropic dysfunction, and in cases the drug causing node dysfunction cannot be interrupted or modified.

In brady-tachyarrhythmia patients, pacemaker can be indicated for symptomatic bradycardia treatment. Anti-arrhythmic drugs are recommended for tachyarrhythmia episode therapy.

Atrial fibrillation or sinus node dysfunction patients, especially those with bradycardia and tachycardia alternation, show high risks for thromboembolic occurrences, even after pacemaker placement[6]. Such correlation may explain previous cerebrovascular accident undergone by the patient.

Facing the diagnosis of acute myocardial infarction with ST segment elevation and 4-hour evolution, the patient was submitted to mechanical coronary reperfusion. Studies support angioplasty option in detriment to thrombolysis based on advantages concerning short- and long-term survival among elderly populations[7].

Patients over 85 years of age have mortality rate three times higher than those under 65, in post-myocardial infarction period[8].

Sudden death evolution on the first post-AMI day provides us with many diagnostic options. Diagnostic death hypotheses include myocardial infarction complications (arrhythmias, ischemic and mechanical), catheterization complications, pulmonary thromboembolism, and even a new cerebrovascular accident.

In-hospital death in acute myocardial infarction patients is caused largely circulatory failure due to severe left ventricular dysfunction or one of acute myocardial infarction complications[9].

Severe left ventricular dysfunction with cardiogenic shock is usual after acute myocardila infarction. Such severity is correlated with infarction extension. Risk factors for cardiogenic shock development are presence of previous myocardial infarction, old age, female gender, diabetes, and anterior wall infarction. Mortality rate is 80% in infarctions evolving to cardiogenic shock[10]. In this case, the most common practice characterizes for pulmonary congestion and low cardiac outcome signs. This patient did not show such features.

Concerning myocardial infarction complications, lethal ventricular arrhythmia must be considered as sudden death cause of this patient. Death due to ventricular tachyarrhythmias in AMI is one of the most frequent death causes, as it represents 90% of those cases[9]. In patients with ST elevation acute myocardial infarction, ventricular arrhythmias incidence is greater than in those with unstable angina or non-ST elevation infarction. Those may be caused by a new ischemic attack or due to previous anatomic changes as old necrosis areas, which served as reentry circuits for electrical stimulation. Regarding this patient, it is important to emphasize that EKG already shows an electrically inactive anteroseptal area and coronary angiography demonstrated dyskinetic area in anterolateral and apical walls, which may correspond to an aneurism area as anatomic substrate for arrhythmias. Epidemiological data demonstrate new ischemic attack as the highest related mechanism to ventricular arrhythmias in acute myocardial infarction. However, presence of pre-established substrate is a more probable cause in this case. Fatal bradyarrhythmia is also included as possible death causes concerning previous sinus node disease diagnosis.

Another fast evolving to death AMI complication would be cardiac rupture. Free wall rupture takes place in about 2 to 3% of patients. However, it is responsible for approximately 10% of in-hospital mortality due to acute myocardial infarction[11]. It is the

third most common post-infarction death cause after cardiogenic shock and ventricular arrhythmias. Left ventricular free wall rupture is 8 to 10 times more frequent than ventricular septum and papillary muscle rupture. It takes place on the first five days in about 50% of patients, and 90% in two post-infarction weeks[12]. Free wall rupture only occurs in transmural infarction. Old age (>70 years old), female gender, hypertension, first myocardial infarction, and absence of collateral circulation[12] are risk factors for left ventricular free wall rupture. In acute rupture, patients show electromechanical dissociation and sudden death. Sudden thoracic pain when coughing may reveal the beginning of cardiac rupture. Some patients may have subacute pattern as result from tamponed rupture with suggestive thoracic pain from pericarditis, nausea, and hypotension[13].

In a large post-infarction retrospective study[13], approximately 6.2% of patients had cardiac rupture and about a third showed subacute disease. There are three types of free wall rupture: a) type I rupture, which happens within the first 24 post-infarction hours and it is a full wall thickness rupture; b) type II rupture occurs as a result from myocardial erosion in infarcted area; and c) type III rupture, which takes place later and it is located on edges around the infarction where normal myocardium is present. The only chance for survival in those cases is immediate diagnosis and emergency surgery.

In this patient case, type I left ventricular free wall rupture or a possible aneurism rupture would justify sudden death due to cardiac tamponade development, and further cardiac arrest due to electromechanical dissociation and asystole, as she showed most risk factors for that complication.

Another mechanical complication of infarction is acute mitral failure occurring more often in inferior wall infarction due to rupture of posteromedial papillary muscle. Such complication takes places in approximately 1% of myocardial infarction patients and is responsible for 7% of cardiogenic shocks. In some cases, it represents 5% of post-myocardial infarction mortality[14]. In approximately 50% of cases, infarction is small and rupture usually takes place between the second and seventh post-infarction day, although some studies show a mean rupture time of about 13 hours[15]. In those cases, the most frequent clinical presentation would be sudden and intensive pulmonary congestion with pulmonary edema or cardiogenic shock development, which did not occur with this patient.

In the extent of infarction-related mechanical complications, we must consider interventricular septum rupture. Such complication is responsible for 5% of peri-infarction mortality. Furthermore, its incidence has considerably decreased after the beginning of early reperfusion[16]. Risk factors are old age, female sex, hypertension, anterior wall infarction, tachycardia, and pulmonary congestion signs at hospital admission[17]. Rupture usually occurs between the second and fifth post-infarction day. However, occurrences within the first 24 hours have been observed[17]. It reveals signs of pulmonary congestion, biventricular failure, and hypotension. Such findings were not verified in our patient.

Finally, death due cardiac catheterization complications is included in differential diagnosis. Myocardial infarction in 0.05%, cerebrovascular accident in 0.07%, arrhythmias in 0.38%, and chamber perforation in 0.28% distinguish among them. Procedure-related mortality is 0.11%[18].

So we have 1) mechanical complication of acute myocardial infarction – heart free wall rupture; 2) Sudden death due to ventricular arrhythmia, as death hypothesis.

(Dr Ariane Veira Scarlatelli Macedo)

Special guest's comments

The case of this 88-year-old woman who died during an acute myocardial infarction is a glimpse of the future of cardiovascular medicine. Demographic trends indicate that the practice of cardiology will increasingly involve the "elderly old", in their eighties and nineties. Increasingly, this segment of our patient population will consist predominantly of women. Aspects of this woman's case are all too common. She had classical coronary risk factors, including advanced age and longstanding hypertension. She had sustained a previous cerebrovascular accident, although we were not told whether this was hemorrhagic or ischemic. Nonetheless, a prior stroke substantially augments for a further cardiovascular complication.

Although typical myocardial infarction in such an individual would not be unusual, certain aspects of this particular case warrant further comment. This woman was taken urgently to the cardiac catheterization laboratory for primary angioplasty. I find this disposition of a woman within the first hours of an acute myocardial infarction very appropriate. I congratulate the emergency and invasive cardiology team for their appropriately aggressive stance. All too often, we underestimate the symptoms of ischemic heart disease in women. Studies in North American have shown a systemic under-recognition of myocardial ischemic symptoms, and less aggressive management of women than men and in older versus younger individuals. The benefits of mechanical revascularization appear evident, although the numbers of individuals randomized in clinical trials in this age stratum are understandably fewer than with younger individuals.

I am sure, however, that the invasive cardiology team was surprised to find angiographically normal-appearing coronary arteries. Dr. Ariane Macedo has already provided us with an extensive differential diagnosis of myocardial infarction with normal-appearing coronary arteries. Noteworthy possibilities include vasospasm, most typical in younger individuals. Embolic causes with lysis of the occluding thrombus by the time of angiography also bear consideration. Bland emboli could be one source of a coronary thrombus. Indeed, this woman had documented sick sinus syndrome with tachybrady syndrome. However, there is no documentation of atrial fibrillation or atrial flutter, arrhythmic conditions associated with cardiac source of emboli. Septic emboli from endocarditis, for example, also could cause acute myocardial infarction. Indeed, this woman's history and left ventriculogram were compatible with significant mitral regurgitation. However, there were no clinical signs of sepsis, rendering this possibility less likely. In North America, an additional consideration for acute myocardial infarction without typical coronary atherosclerosis would be cocaine use. That diagnosis probably does not pertain to the current case.

However, we always query patients with acute myocardial infarction about substance abuse. Another cause of acute myocardial infarction with extensive distal wall-motion abnormalities on left ventriculogram, the Takotsubo syndrome, also can cause myocardial infarction with angiographically normal-appearing coronary arteries. Typically, the electrocardiogram shows more pro-

minent T-wave inversion and the ST segment elevation with reciprocal ST segment depression so well described by Dr. Paulo Moffa in his discussion of the electrocardiogram. In addition, the Takotsubo syndrome usually is precipitated by a severe emotional shock. There is no evidence for this in the case at hand. Although Takotsubo syndrome affects women much more often than men, these women tend to be much younger than 88 years old. Finally, rendering the diagnosis of Takotsubo syndrome highly unlikely, the prognosis is excellent with recovery of ventricular function, rather than rapid progression to death, as in the present case. Dr. Ariane Macedo raised the possibility of infiltrative cardiomyopathy. Given the focal nature of the electrocardiographic changes, and the regionality of the extensive left ventricular dysfunction noted on the left ventriculogram by Dr. Eulogio Martinez, I consider infiltrative disease much less likely as an explanation for this patient's current clinical scenario.

In the presence of traditional coronary risk factors, I believe that the likely explanation for the acute myocardial infarction in this woman is atherosclerotic coronary artery disease. We have learned over the last decade or so that many acute myocardial infarctions result from a physical disruption of atherosclerotic plaques that do not necessarily cause a critical coronary artery stenosis. We also have learned that the coronary arteriogram vastly underestimates the extent of coronary atherosclerosis.

Traditionally thought of as a segmental disease, classical autopsy studies and contemporary intravascular ultrasound studies show the ubiquity of intimal disease in individuals with coronary atherosclerosis. The lesions usually occur much more diffusely than the coronary angiogram would suggest. The Glagov phenomenon, compensatory enlargement or outward remodeling, protects the caliber of the lumen of the coronary artery affected by atherosclerosis. It is not until the late stage of this disease that luminal encroachment visible on the angiogram occurs. A disruption with thrombosis of a plaque at a site of outward remodeling of the artery, followed by lysis or distal embolization of the thrombus into the microcirculation after fragmentation could explain the apparently normal coronary arteriogram in the face of the documented myocardial infarction in this woman. I prefer use of the cumbersome term "angiographically normal-appearing coronary arteries" to the shorter but all too often misleading term "normal coronary arteries" to describe the cinecoronariographic findings, to remind us of the limitations of luminography in gauging atherosclerosis, as disease of the artery wall that only occasionally narrows the lumen.

The other particular feature of this clinical scenario that bears comment is the cause of death. Presumably, this woman who succumbed within the first day of an acute myocardial infarction was in a monitored ward. There is no mention of a brady- or tachyarrhythmia as a primary complication of her myocardial infarction that led to her death. We are not told of a downward hemodynamic spiral preceding the terminal event that would suggest primary pump failure or cardiogenic shock in the woman with severe impairment of left ventricular function. While the case protocol is not explicit, the clinical scenario of sudden, unheralded cardiac arrest and a prolonged resuscitative effort evokes the clinical scenario now called pulseless electrical activity cardiac arrest (PEA), previously referred to as electromechanical dissociation. In patients in the acute phase of myocardial infarction, I agree with Dr. Ariane Macedo that cardiac rupture should be considered in such circumstances. Dr. Paulo Moffa described low voltage in the frontal plane on the electrocardiogram. This could indicate pericardial effusion. However, this electrocardiographic finding preceded by some time the abrupt death and could well be unrelated to a hemopericardium secondary to cardiac rupture. However, as Dr. Ariane Macedo pointed out, this woman had many risk factors for cardiac rupture, including longstanding hypertension, female gender, advanced age, and no history of prior myocardial infarction. While myocardial ruptures typically occur later than the first day after onset of infarct symptoms, the clinical scenario strongly suggests consideration of this cause of death.

(Dr. Peter Libby)

Necropsy

At necropsy, partially hemorrhagic myocardial infarction, in left ventricular anterior wall and its adjacent portions, septum and right ventricular anterior wall, was the main finding. Myocardial rupture took place in those last two locations, with interventricular communication and blood overflowing to pericardial sac (figure 5). Death was due to cardiac tamponade. Infarction evolution time can be estimated by morphologic criteria, such as approximately a day (figure 6).

Coronary arteries showed mild blockage due to atherosclerosis (up to 69% at left coronary circumflex branch and 77% at right coronary artery). Non-occlusive mural thrombus in anterior interventricular branch anterior established 82% obstruction (figure 7) with atherosclerosis.

Sinus node fibrosis (figure 8) was shown by microscopic study. That change is responsible for chronic cardiac rhythm adjustments the patient showed, and ischemic disease is its possible cause. Amyloidosis was not even found in atrial septum, which is very usual among elderly individuals.

Myocardial left ventricular hypertrophy, secondary to systemic blood pressure, was also present. Benign renal atherosclerosis was detected.

(Dr. Paulo Sampaio Gutierrez)

Anatomopathological Diagnoses

Main disease - Myocardial infarction without total coronary artery occlusion.

Causa mortis - Cardiac tamponade due to septum and right ventricle rupture.

(Dr. Paulo Sampaio Gutierrez)

Comment

Coronary thrombosis, usually occlusive and secondary to atherosclerosis plaque rupture, is the most common myocardial infarction cause. The most prone to rupture plaque usually has high lipidic content and is responsible for severe coronary artery obstruction, at least at histopathological study (although it is not possible at the coronary angiographic one). However, it did not take place in the present case, in which recent thrombus was not present, despite the patient had died on the first post-infarction day. Two possibilities must be taken into consideration in trying to explain myocardial ischemic necrosis. The most likely is it has been due to vasospasm,

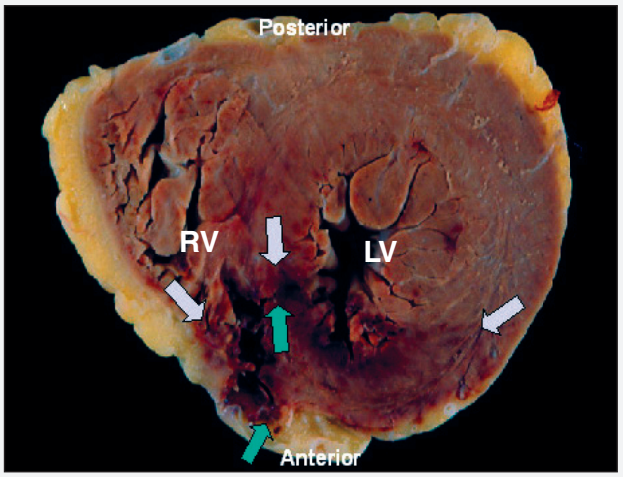

Figure 5 - Heart cross section at ventricle level; posterior portion corresponds to upper part in photograph. Partially hemorrhagic myocardial infarction, limited by with arrows, is observed. Green arrows indicate ventricular rupture in necrotic region. RV - right ventricle; LV - left ventricle.

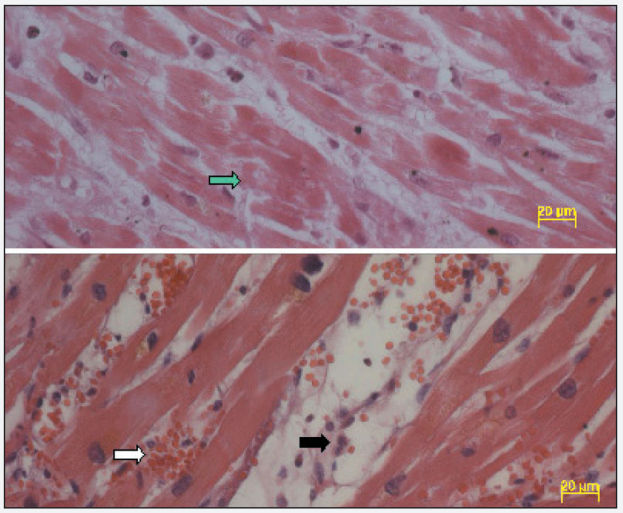

Figure 6 - Septal myocardium histological section showing current necrosis area in contraction sides (illustrated by the green arrow), as well as neutrophil areas (some of which indicated by the black arrow) and erythrocytes (indicated by the white arrow), indicating necrotic area hemorrhage. (Hematoxylin and eosin; objective lens augmentation - 40x).

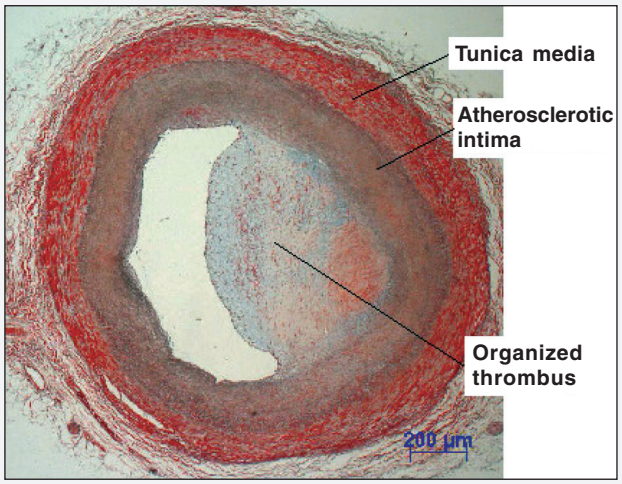

Figure 7 - Cross section photomicrograph of anterior interventricular branch first centimeter (anterior descending) of left coronary artery. Fibrotic atherosclerotic lesion and organized non-occlusive thrombosis are present. (Movat method coloring; objective lens augmentation - 2,5x).

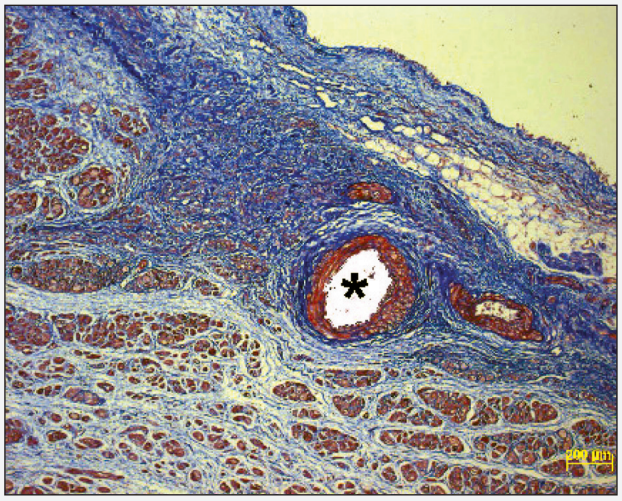

Figure 8 - Heart photomicrograph around sinus node artery (marked with asterisk), displaying long myocardial substitution zones (in red) for fibrosis (in blue). (Masson method coloring; objective lens augmentation - 5x).

which usually affects atherosclerotic lesion arteries with less severe occlusion, such as this patient's. Instead, infarction must have been caused by thrombus embolia found in anterior interventricular branch. Lesion hemorrhage confirms that after blood flow interruption, which caused myocardial fiber demise, it was reestablished. That reinforces the possibility of occurrence of vasospasm. If embolia had led to occlusion, it should have been fixed.

In a recent report[19], we showed 14 cases of cardiac tamponade present in necropsies carried out at Laboratório de Anatomia Patológica (Pathological Anatomy Laboratory) of Instituto do Coração between 2000 and 2004. Mean age of necropsy-submitted patients who suffered myocardial rupture was 70.1 years old and median 74, with prevalence of men (55%). Rupture took place, in average, on day 6.6 from infarction evolution (median 4). First infarction rupture occurred in 72.2% of cases; hypertension was diagnosed in 77.8%, and diabetes mellitus in 27.8% of cases.

Table I adds data from 4 other cases: two with papillary muscle rupture and two with interventricular communication, without simultaneous lesion of right ventricle. Most patients are male. However, if we consider there is prevalence of men in total necropsies, it is observed the complication affecting proportionally more women than men. Likewise in other series[20,21], in 13 (72.2%) from 18 cases it corresponded to first infarction, as well as in systemic hypertension patients. No relation has been found between infarction size and rupture risk[20]. It is no rare the infarction is small.

Dual rupture was found in the present case and in another from our series. It is an unusual form of complication, but previously reported[21,25]. When indicated in those studies, dual rupture proportion in total cases with that complication was similar to ours (2/18=11.1%), varying from 7.7% to 12.9%[21,22,25]. On the other hand, few are the cases described in which rotten free wall has been right ventricular[24,25].

(Dr. Paulo Sampaio Gutierrez)

Table I - Demographic, clinical, and morphologic characteristics observed in necropsies of myocardial rupture patients

Age/sex	Rupture location	Ventricular wall	Days	Hypertension	Diabetes mellitus	Treatment
442/male	LV free wall	Anterior, lateral, and posterior	3	-	-	Thrombolysis
449/male	LV free wall	Lateral	23	+	-	Angioplasty
553/femiminio	Papillary muscle	Anterior and lateral	14	+	-	-
558/male	Interventricular comm..	Septum	14	+	+	-
664/male	LV free wall	Lateral	5	-	-	-
665/female	LV free wall	Lateral, anterior, and posterior	4	+	-	-
669/female	LV free wall	Lateral	10	+	-	Surgical revascularization
772/female	LV free wall	Posterior	4	+	+	Thrombolysis and surgical revascularization
773/male	Interventricular communication, LV free wall and RV	Septum and right ventricle				
775/male	Papillary muscle	Lateral and Posterior	2	-	+	Previous surgical revascularization
777/male	Free wall	Lateral	4	+	-	-
777/male	LV free wall	Lateral	4	+	-	Previous surgical revascularization and angioplasty
778/male	LV free wall	Lateral	2	+	-	-
779/female	LV free wall	Posterior	7	-	-	-
880/male	Interventricular communication	Lateral, anterior, and septal	14	+	-	-
882/female	LV free wall	Lateral and posterior	5	+	-	Thrombolysis
882/female	LV free wall	Lateral	1	+	+	-
888/female	Interventricular communication, LV free wall, RV	Septum and right ventricle	2	+	-	-

LV - left ventricle; RV - right ventricle.

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