Case 4/2014 - A 66-Year-Old Man with Acute Myocardial Infarction and Death in Asystole after Primary Coronary Angioplasty

Wilma Noia Ribeiro, Alice Tatsuko Yamada, Luiz Alberto Benvenuti

Instituto do Coração (InCor) HC-FMUSP, São Paulo, SP - Brazil

A 66-year-old man sought medical care at the hospital due to severe chest pain lasting for 24 hours. The patient was aware of being hypertensive and was a smoker. Without any prior symptom, he started to have severe chest pain and sought emergency medical care after about 24 hours, due to pain persistence.

At physical examination (August 13, 2005, 10 PM) he had a heart rate of 90 bpm and blood pressure of 110/70 mmHg. Lung examination showed no alterations. Heart assessment showed a systolic murmur in the lower left sternal border and mitral area.

The initial electrocardiogram (August 13, 2005, 22 h) showed HR of 100 bpm, sinus rhythm, 1st-degree atrioventricular block (PR 240 ms), low-voltage QRS complexes in the frontal plane, QRS complex electrical alternans and extensive ongoing anterior wall infarction (QS V1 to V6, ST elevation in the same leads and QS in the inferior wall, II, III and aVF) (Figure 1).

Acetylsalicylic acid by oral route and 5 mg of intravenous metoprolol were administered. The patient had bradycardia and cardiorespiratory arrest in pulseless electrical activity, reversed after five minutes. He developed hypotension and peripheral hypoperfusion and was transferred to InCor (The Heart Institute).

On admission he had received heparin and continuous intravenous norepinephrine. BP was 60/30 mmHg.

The ECG (August 13, 2005, 11:36 PM) disclosed heart rate of 116 bpm, junctional escape rhythm with sinus arrest and atrial extrasystoles); low-voltage QRS complex in the frontal plane, extensive ongoing anterior acute myocardial infarction, inactive area in the inferior wall; presence of ST elevation at V1 to V5 and ST depression in leads I, II and aVF; ST elevation in aVR (Figure 2).

Coronary angiography was indicated, which disclosed anterior interventricular branch occlusion and images suggestive of intracoronary thrombus, lesion of 70% in the circumflex artery, 50% in the right coronary artery and 70% in the ostium of the right posterior descending branch. Angioplasty was performed with stent implant in the anterior interventricular artery, but distal flow was not restored. This was followed by cardiac arrest in asystole, which did not respond to treatment and the patient died.

Keywords

Myocardial Infarction; Heart Arrest; Angioplasty, Balloon, Coronary.

Section Editor: Alfredo José Mansur (ajmansur@incor.usp.br)

Associated Editors: Desidério Favarato (dclfavarato@incor.usp.br)

Vera Demarchi Aiello (anpvera@incor.usp.br)

Mailing Addres: Vera Demarchi Aiello •
Avenida Dr. Enéas de Carvalho Aguiar, 44, subsolo, bloco I, Cerqueira César.
Postal Code 05403-000, São Paulo, SP – Brazil
E-mail: demarchi@cardiol.br, vera.aiello@incor.usp.br

DOI: 10.5935/abc.20140129

Clinical Aspects

This clinical case reports on a 66-year-old hypertensive patient, long-term smoker, who sought medical care due to acute chest pain. The main diagnostic hypothesis for this clinical case is of acute coronary syndrome.

Chest pain is one of the most common reasons for seeking emergency care and remains a challenge for the clinician, due to the difficulty in differentiating between non-emergency diagnoses and those of high morbidity and mortality, such as acute coronary syndrome (ACS), aortic dissection and pulmonary thromboembolism.

In the assessment of acute chest pain, there are three basic parameters for its management: clinical examination (clinical history and physical examination), electrocardiogram (ECG) and myocardial necrosis markers. They should be analyzed together to provide a safer approach to the patient, especially when it is necessary ruling out ACS. Chest radiography, chest Computed Tomography Angiography (CTA), echocardiography and other tests may be useful in the differential diagnosis.

Approximately 15-25% of patients presenting in the ER with chest pain are diagnosed with acute coronary syndrome, and this represents its more frequent clinical manifestation[1]. Therefore, in the first step of the evaluation, which is the clinical examination, the greater determinant of an ischemic etiology is the characteristic presence of angina.

Angina is often described as a burning or compression sensation or difficulty breathing, located in the precordial region or any other region of the chest, radiating to the neck, shoulder and left arm. It usually increases in intensity within minutes and may be accompanied by symptoms such as nausea and sweating. It can be triggered by physical or emotional stress and relieved by rest or use of nitrates. It should also be remembered that ACS can occur without obvious precipitating factors and be asymptomatic or present as ischemic equivalent, especially in the elderly and diabetic patients with autonomic dysfunction (dyspnea, syncope and pre-syncope).

On the other hand, there are characteristics of pain that make the diagnosis of ACS unlikely, such as pleuritic pain (reproduced by respiratory movements) located with the fingertip, pain in meso/hypogastrium region and reproduction of pain with local

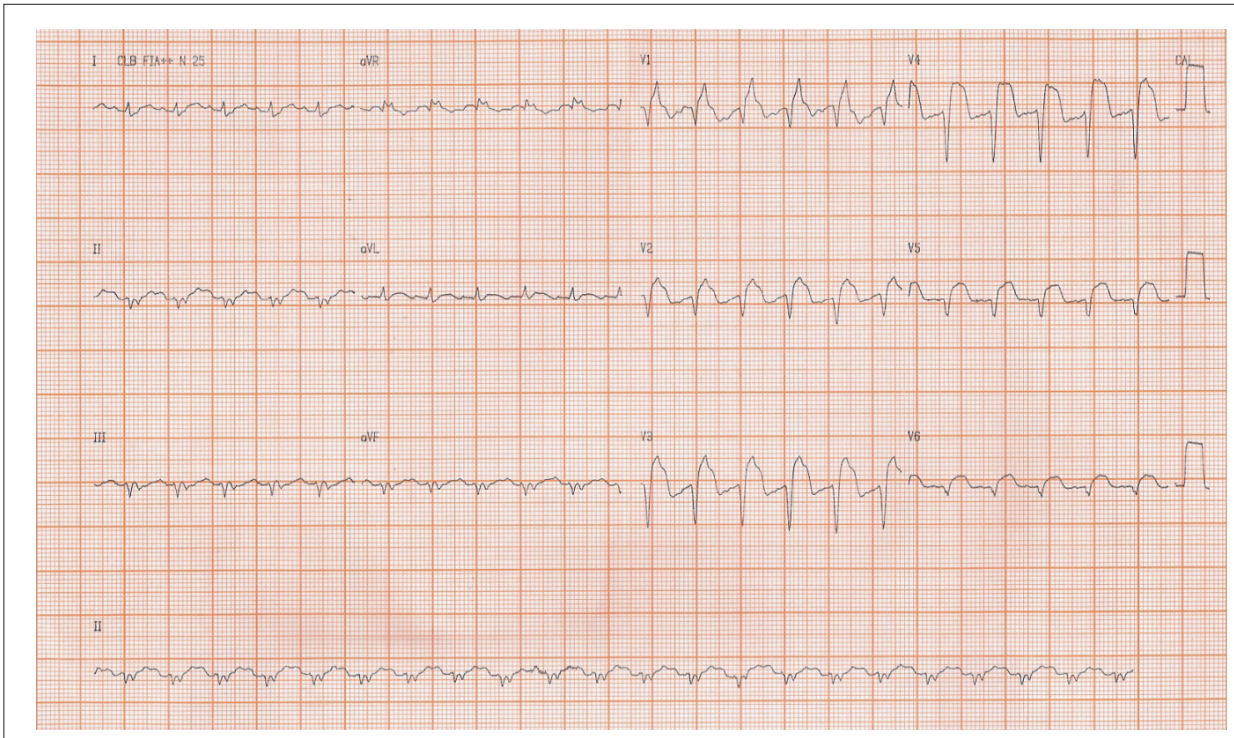

Figure 1 – *ECG: low QRS voltage in the frontal plane, electrical alternans of QRS complexes, electrically inactive lower wall area and extensive ongoing myocardial infarction.*

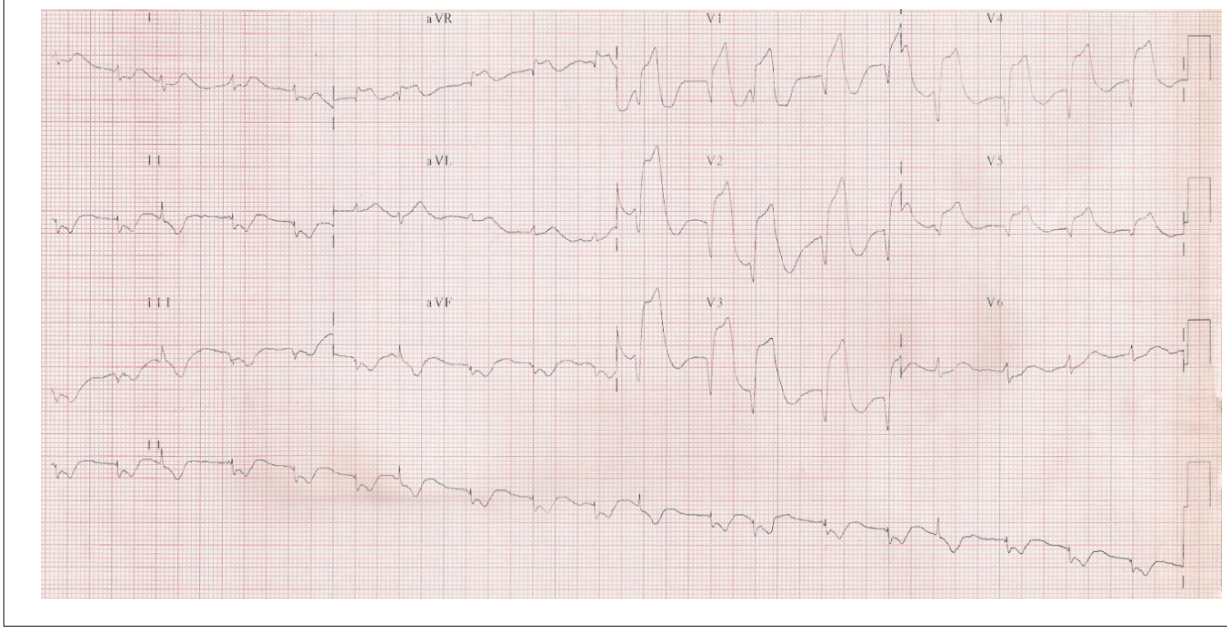

Figure 2 – *ECG: low QRS voltage in the frontal plane, electrically inactive lower wall area and anterior myocardial infarction with increased ST elevation, still with positive T waves, "hyperacute phase of myocardial infaction".*

Anatomopathological Session

palpation or movement. These features raise the suspicion of other differential diagnoses such as pericarditis, pleuritis, gastrointestinal or musculoskeletal diseases.

In the present case report, the patient presented with prolonged chest pain, which does not rule out acute coronary syndrome (ACS), but raises the possibility of some condition associated with this coronary picture, such as pericarditis or mechanical complications.

Among the most important risk factors for atherosclerotic disease risk are dyslipidemia, diabetes mellitus, hypertension, male gender, older age, obesity/metabolic syndrome, smoking, sedentary lifestyle, chronic kidney disease, depression and stress. This patient had some risk factors that contributed to the development of coronary artery disease: age, male gender, hypertension and smoking.

Patients with chest pain and ACS often have a nonspecific physical examination, with less than 20% of them showing significant alterations in the initial evaluation[2]. This becomes important by helping in the detection of differential diagnoses (e.g., pericardial friction rub in pericarditis) or by inferring the presence of risk factors for coronary artery disease (abdominal or carotid murmur, among others).

However, when findings resulting from an ACS are present, they indicate a worse prognosis due to mechanical complications or due to a large area of myocardium at risk and ventricular dysfunction (hypotension, tachycardia, pulmonary edema and mitral regurgitation murmur secondary to ischemia).

The electrocardiogram is important in the diagnostic, prognostic and therapeutic approach and must be obtained within 10 minutes after the presentation of patients with ongoing chest pain[2]. A normal electrocardiographic tracing does not exclude the possibility of ACS and a serial ECG is indicated, which increases its sensitivity and helps differentiating between acute and chronic alterations.

The patient reported in this clinical case had, at the admission ECG performed at another service, ST-segment elevation in the anterior wall, suggesting the hypothesis of acute coronary syndrome with ST-segment elevation. However, this ECG also showed low voltage and electrical alternans of the QRS complex, which suggests large pericardial effusion or even cardiac tamponade.

The main hypothesis for this pericardial effusion is a mechanical complication of myocardial infarction: left ventricular free wall rupture. It occurs within 24 hours after infarction or between the third and fifth day, has an incidence of 0.8 to 6.2% and is more common in an extensive myocardial infarction, in the elderly, women and hypertensive patients. Its clinical course is variable[3] and may be acute and severe, leading to sudden death or subacute, with nonspecific clinical manifestations.

Other mechanical complications that may be present in myocardial infarction are papillary muscle and interventricular septum rupture. These complications do not present with significant pericardial effusion and normal pulmonary auscultation in this patient also makes the diagnosis of papillary muscle rupture less likely. This clinical condition presents with pulmonary congestion due to volume overload secondary to acute mitral regurgitation.

Another diagnostic hypothesis for this patient presenting with chest pain and pericardial effusion would be aortic dissection. Pain, in these cases, is usually of sudden onset and strong intensity since the beginning (unlike angina pain, which often increasingly escalates). It is often described as excruciating and its location reflects the site and progression of the dissection. Autonomic signs (pallor, profuse sweating) are greatly associated.

In aortic dissection, physical examination may disclose hypertensive crisis, differences between limb pulses, signs of pleural and pericardial effusion, diastolic murmur of aortic regurgitation, different from the systolic murmur detected in this clinical case. The extension of the dissection to other vessels can lead to other symptoms corresponding to ischemia of the organs irrigated by them: cerebrovascular accident, acute myocardial infarction, mesenteric ischemia, etc.

Another diagnostic hypothesis for the clinical case is pulmonary embolism. The absence of pulmonary symptoms, mainly dyspnea, makes this hypothesis less likely. It is the most common symptom of this disease, occurring in over 78% of the patients[4]. Sudden chest pain of sudden onset and very often pleuritic, affects up to 44% of patients[4]. Cough and hemoptysis may also occur. Additionally, there was no mention is made on admission at the other service, of right ventricular dysfunction manifestations, such as jugular stasis and hypotension.

The patient, an hour and 36 minutes after his admission at the Heart Institute, was submitted to coronary angiography with left anterior descending artery angioplasty. However, he developed asystole and cardiac arrest.

The main diagnoses for the final clinical picture are cardiogenic shock and/or distributive shock due to cardiac tamponade, discussed below.

The hypothesis of cardiogenic shock should be considered, as the patient had an extensive acute myocardial infarction without culprit artery reperfusion even after percutaneous revascularization attempt. However, this diagnosis cannot fully explain the patient's clinical condition, such as the absence of pulmonary congestion, which usually follows an acute myocardial failure.

Considering the patient's history, late cardiac tamponade seems to have been the main precipitating factor of the final clinical picture in this case. The electrocardiographic findings commonly observed in cardiac tamponade are low voltage and electrical alternans of the QRS complex, observed in the case. Although physical examination made no reference to clinical findings suggestive of tamponade, such as jugular stasis or muffled heart sounds, we cannot exclude this diagnostic hypothesis.

An echocardiography could have been performed to confirm this diagnosis, which is the most widely used noninvasive method for diagnostic investigation of this pathology. The ventriculography in this context would not be informative, as it was a free wall rupture with cardiac tamponade and thus, it would not allow the visualization of contrast leakage into the pericardial cavity.

This is a patient with myocardial infarction that came at the emergency room more than 24 hours after the onset of the event and who probably had a mechanical complication of myocardial infarction: ventricular free wall rupture.

Most deaths from myocardial infarction occur in the first hours of disease onset, with 40-65% occurring within the first hour and approximately 80% in the first 24 hours[5,6]. The recently implemented therapies for MI treatment have been proven to modify patient evolution and prognosis. However, the effectiveness of most of these measures is time-dependent and delay in seeking medical care may have been the factor that likely contributed to the clinical outcome of the patient in this case report. **(Dr. Wilma Noia Ribeiro, Dr. Alice Tatsuko Yamada)**

Diagnostic hypotheses: Acute myocardial infarction with mixed shock (cardiogenic - distributive) by mechanical complication - free wall rupture with tamponade **(Dr. Wilma Noia Ribeiro, Dr. Alice Tatsuko Yamada)**

Necropsy

The heart weighed 414 g. The myocardium of the left ventricular anterosseptal wall and right ventricular anterior wall was softened, slightly yellowish in color, characterizing extensive transmural acute myocardial infarction. There was an obvious narrowing of the affected anterosseptal wall, with ventricular septum rupture in the anterior region of its mid portion, with a ventricular septal defect measuring 10 mm in its longest axis. The other left ventricular walls showed to be slightly hypertrophic and there was a small area of fibrosis in the postero-inferior region of the ventricular septum. There was also moderate right ventricular dilation (Figure 3).

Histological analysis confirmed the presence of myocardial infarction, with marked neutrophil infiltration, confirming histological dating of 24-48 hours of onset. Another small ongoing microinfarction was observed in the posterior region of the ventricular septum, in addition to the previously described small area of fibrosis (healed infarction), compatible with approximately 7-10 days of evolution.

There was superficial fibrin deposition in the epicardium, with the presence of reactive inflammatory infiltrate. Examination of the initial segment of the left anterior descending artery showed fatty atherosclerotic plaques with areas of marked thinning of the fibrous cap that covered the lipid core and 80% of obstruction.

There were also areas of plaque rupture and hemorrhage, with acute thrombosis in the first and second centimeters of that artery (Figs. 4 and 5).

The lungs weighed 1,208 g together and showed alveolar edema. The kidneys showed irregular surface and retention cysts, with hyaline arteriolosclerosis on histological examination. The aorta showed mild / moderate degree of atherosclerosis. **(Dr. Luiz Alberto Benvenuti)**

Anatomopathological diagnoses. Coronary atherosclerosis; acute myocardial infarction involving the left ventricular anterosseptal wall and the right ventricular anterior wall; rupture of the ventricular septum, with VSD; acute pulmonary edema (cause of death) **(Dr. Luiz Alberto Benvenuti)**.

Comments

This is the case report of a 66-year-old man with systemic hypertension and a chronic smoker that presented with acute severe chest pain. After medical assessment, he was diagnosed with acute myocardial infarction and the patient underwent

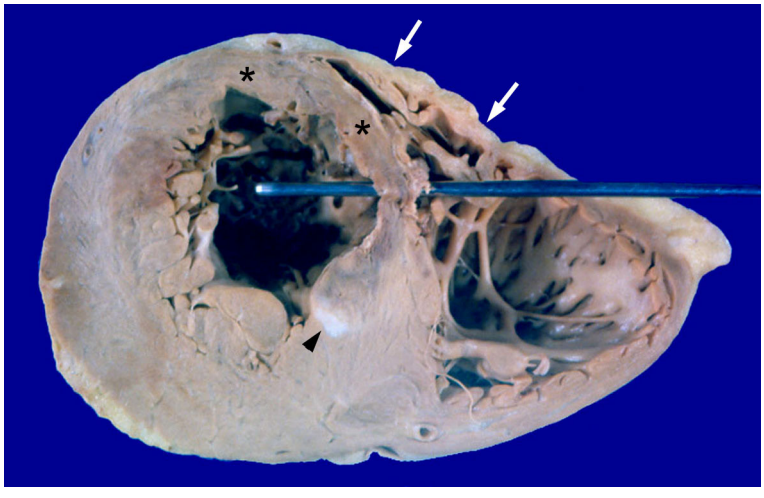

Figure 3 – *Cross-section of the ventricles showing left ventricular transmural infarction of the anterosseptal wall (asterisks) and of the right ventricular anterior wall (arrows). The explorer shows the VSD secondary to septal rupture. Observe the ventricular wall thinning and the small area of fibrosis in the ventricular septum (arrowhead).*

Anatomopathological Session

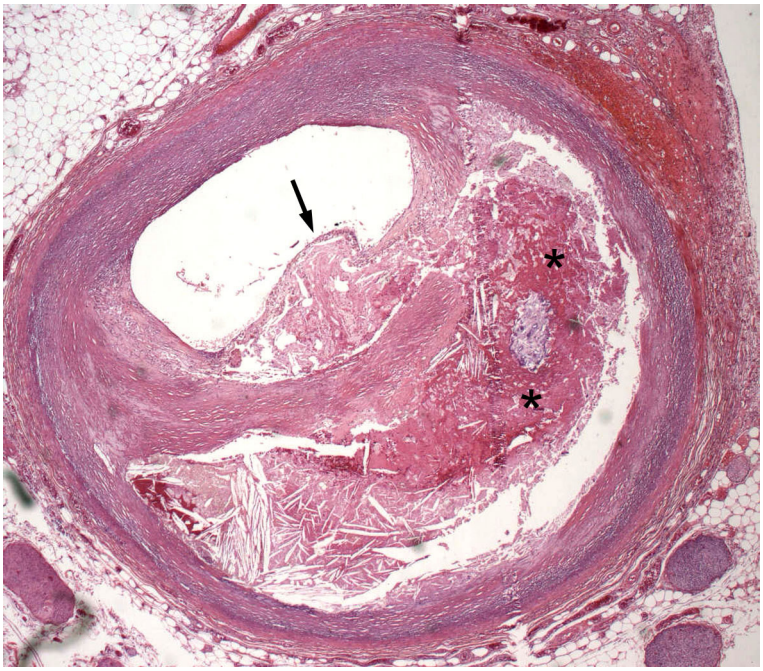

Figure 4 – *Histological section of the first centimeter of the left anterior descending artery showing large lipid-core atherosclerotic plaque, with internal area of fibrin deposition and hemorrhage (asterisk). Observe the area with marked thinning of the fibrous cap of the lipid core plaque (arrow), site of potential rupture and thrombosis. Hematoxylin-eosin, 2.5×.*

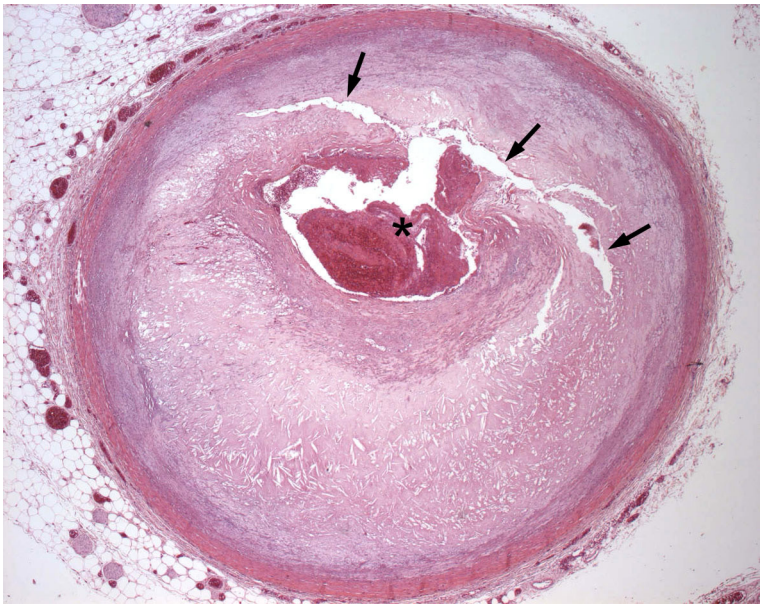

Figure 5 – *Histological section of the second centimeter of the anterior interventricular artery showing large lipid-core atherosclerotic plaque with ruptured area (arrows) and occlusive luminal thrombosis (asterisk). Hematoxylin-eosin, 2.5×.*

coronary angiography, which disclosed proximal occlusion of the left anterior descending artery with images suggesting the presence of thrombi. He was submitted to balloon-angioplasty in the affected segment without restoration of distal coronary flow (unsuccessful procedure) and the patient developed irreversible cardiac arrest and died.

The autopsy confirmed acute myocardial infarction, which was very extensive, affecting the left ventricular anterosseptal wall and the right ventricular anterior wall. Histological dating was 24-48 hours of onset, consistent with the clinical history. It is noteworthy the fact that the detailed examination of the ventricular septum showed the presence of two previous microinfarctions, an old (healed) one and an ongoing one.

The presence of atherosclerosis of the coronary arteries was identified, with massive plaques in the proximal segment of the left anterior descending artery, which resulted in chronic obstruction of 80% of the lumen. The fatty plaques had extensive lipid cores and there were areas of marked thinning of the fibrous cap that covered the cores, as well as areas of rupture associated with acute thrombosis of the remaining lumen in the first two centimeters of the left anterior descending artery. It is known that acute coronary occlusions with luminal thrombosis are usually associated with large lipid-core plaques, which undergo rupture due to the instability of their thin fibrous cap[7], as observed in this case.

Aside from the great extent of the infarcted area, the patient developed an important mechanical complication of acute myocardial infarction, the occurrence of ventricular septal rupture with the establishment of VSD[8] - which certainly aggravated his hemodynamic condition, progressing to cardiogenic shock -, acute pulmonary edema and death. It should be emphasized that the patient had two classic risk factors for atherosclerosis and myocardial infarction: systemic hypertension and chronic smoking[9]. **(Dr. Luiz Alberto Benvenuti)**

References

1. Pope JH, Aufderheide TP, Ruthazer R, Woolard RH, Feldman JA, Beshansky JR, et al. Missed diagnoses of acute cardiac ischemia in the emergency department. N Engl J Med. 2000; 342(16):1163-70.
2. Piegas LS, Feitosa G, Mattos LA, Nicolau JC, Rossi Neto JM, Timerman A , et al. Sociedade Brasileira de Cardiologia. Diretriz da Sociedade Brasileira de Cardiologia sobre tratamento do infarto agudo do miocárdio com supradesnível do segmento ST. Arq Bras Cardiol. 2009; 93(6 supl.2):e179-e264.
3. Becker RC, Gore JM, Lambrew C, Weaver WD, Rubison RM, French WJ, et al. A composite view of cardiac rupture in the United States National Registry of Myocardial Infarction. J Am Coll Cardiol. 1996; 27(6):1321-6.
4. Miniat M, Prediletto R, Formichi B, Marini C, Di Ricco G, Tornelli L, et al. Accuracy of clinical assesment in the diagnosis of pulmonary embolism. Am J Respir Crit Care Med. 1999; 159(3):864-71.
5. Kannel WB, Cupples LA, D'Agostino RB. Sudden death risk in overt coronary heart disease: the Framingham Study. Am Heart J. 1987; 113(3):799-804.
6. Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, Arveiler D, Rajakangas AM, Pajak A. Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. Circulation. 1994; 90(1):583-612.
7. Fleg JL, Stone GW, Fayad ZA, Granada JF, Hatsukami TS, Kolodgie FD, et al. Detection of high-risk atherosclerotic plaque: report of the NHLBI Working Group on current status and future directions. JACC Cardiovasc Imaging. 2012; 5(9):941-55.
8. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med. 2002; 347(18):1426-32.
9. Landini L, Leone A. Smoking and hypertension: effects on clinical, biochemical and pathological variables due to isolated or combined action on cardiovascular system. Curr Pharm Des. 2011; 17(28):2987-3001.