Case 02/2015 – A 67 Year-Old Woman with Sudden Cardiogenic Shock in the 7th Day after Acute Myocardial Infarction

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A 67-year-old woman was sent for a coronary angiography seven days after infarction. The patient presented with prolonged chest pain and was admitted to another hospital because of the diagnosis of acute myocardial infarction.

The first electrocardiogram (ECG) (performed on May 31, 2005 at 4:40 p.m.) revealed no alterations indicating infarction (Figure 1); however, the second ECG (performed on May 31, 2005 at 9:45 p.m.) when a new pain episode occurred revealed alterations indicating infarction.

The second ECG revealed sinus bradycardia, pathological Q waves in leads II, III, and aVF, with ST elevation and positive T waves in the same leads as well as ST depression in V_1 and V_2; these findings are compatible with an acute infarction in progress (Figure 2).

The patient reported less pain and was in Killip Class I.

An ECG (performed on June 7, 2005) revealed pathological Q waves in leads II, III, and avF, with slight ST elevation and T waves that were still positive. The exam also revealed ST depression in V_1 and V_2 (Figure 3).

This same ECG showed that the aorta was 27 mm in diameter, left atrium was 39 mm in diameter, left ventricular diastole was 61 mm in diameter, ejection fraction was 45%, and septal and posterior wall thickness was 9 mm. The left ventricle exhibited apparent diffuse hypokenesis and impaired ventricular filling (E < A); the valves did not show any abnormalities.

On physical examination (performed on June 8, 2005) the patient was in a poor condition overall; she was pale and hypotensive, with cold limbs and increased pressure in the jugular vein. The results of pulmonary and cardiac auscultation were normal; there were no signs of deep vein thrombosis in the legs.

Laboratory tests (performed on June 8, 2005) revealed the following values: hemoglobin 10.8 g/dL, hematocrit 33%, MCV 70 μm^3, leukocytes 9000/mm^3, platelets 245000/mm^3, urea 50 mg/dL, creatinine 1.7 mg/dL, troponin I 32.6 ng/mL (8:00 a.m.) and 27.1 ng/mL (4 p.m.), and CK-MB 3.70 ng/mL (8:00 a.m.) and 4.2 ng/mL (4:00 p.m.).

A coronary angiography performed on June 8, 2013 revealed proximal occlusion of the right coronary artery, left coronary trunk free of obstructive lesions, left circumflex artery free of lesions, and anterior interventricular artery with 70% obstruction in the mid segment. A right coronary angioplasty was performed and showed thrombi; many stents were inserted between the proximal and distal portions.

After the coronary angiography, the patient developed severe dyspnea and respiratory insufficiency. Orotracheal intubation was required for respiratory support, and the patient remained in shock.

The ECG (performed on June 8, 2005) revealed new ST elevation in the inferior wall, with negative T waves and ST depression in V_2 and V_3, aVL, and lead I (Figure 4).

The patient remained in shock with the use of vasoactive drugs and went into cardiorespiratory arrest. She did not respond to resuscitation efforts and died a few hours later.

Clinical Details

This is a case of a 67-year-old woman who presented with an initial episode of chest pain with no obvious alterations indicating ischemia in her ECG. Five hours later, she experienced more pain; this time the pain was accompanied with ST elevation in leads II, III, and aVF, and was diagnosed to be acute myocardial infarction with ST elevation in the inferior wall.

Epidemiological studies show that acute coronary syndrome has high rates (approximately 30%) of overall mortality; half of these deaths occur in the first two hours after the event, and 14% of patients die before receiving medical attention[1]. According to data from the Brazilian Ministry of Health, 27,595 deaths resulting from ischemic heart disease in patients older than 40 years were reported in 2011 alone[2].

Although infarction of the inferior wall is associated with lower rates of morbidity and mortality, the situation cannot be underestimated as in this case, wherein the patient reached Killip I and eight days after admission presented hypotension with increased pressure in the jugular vein and later died. Reinfarction or delayed complications of infarction (such as left ventricular free wall rupture or rupture of the ventricular septum) should be considered as immediate diagnostic hypotheses.

A finding that favors the reinfarction hypothesis can be seen in the echocardiogram performed the day before, which did not show hypokinesia or left ventricle dysfunction that would explain the clinical presentation at that time. The absence of

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Myocardial Infarction; Shock, Cardiogenic; Heart Diseases/complications; Cineangiography/mortality.

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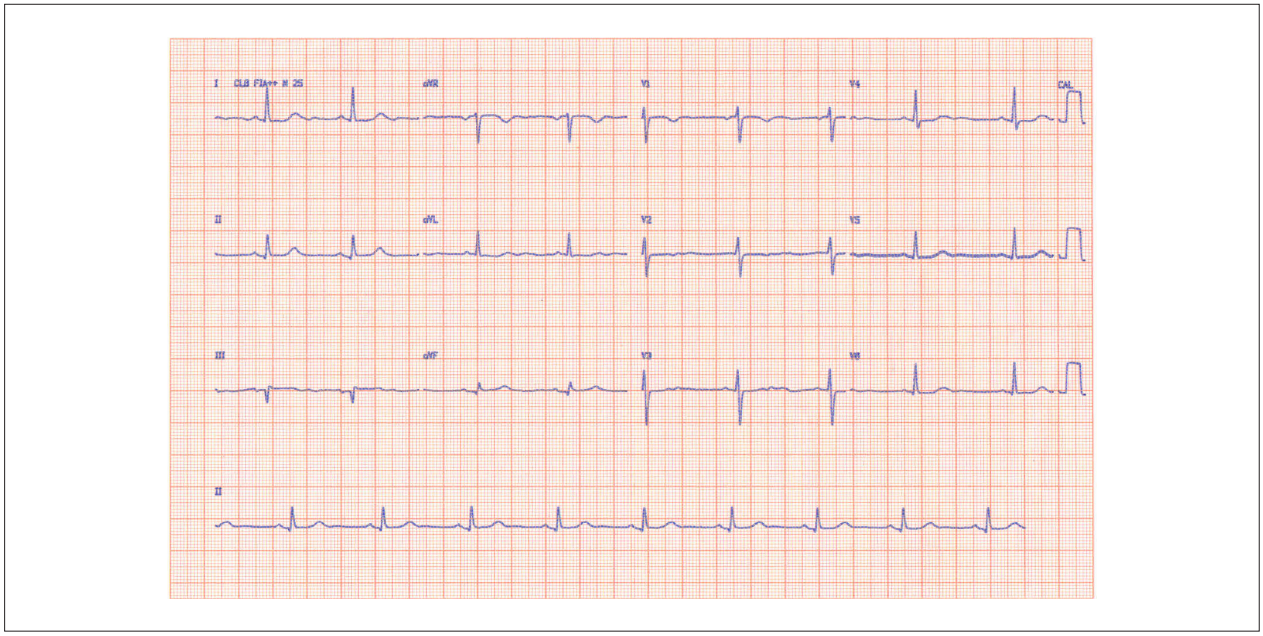

Figure 1 – *ECG: sinus rhythm, likely electrically inactive area of the inferior wall.*

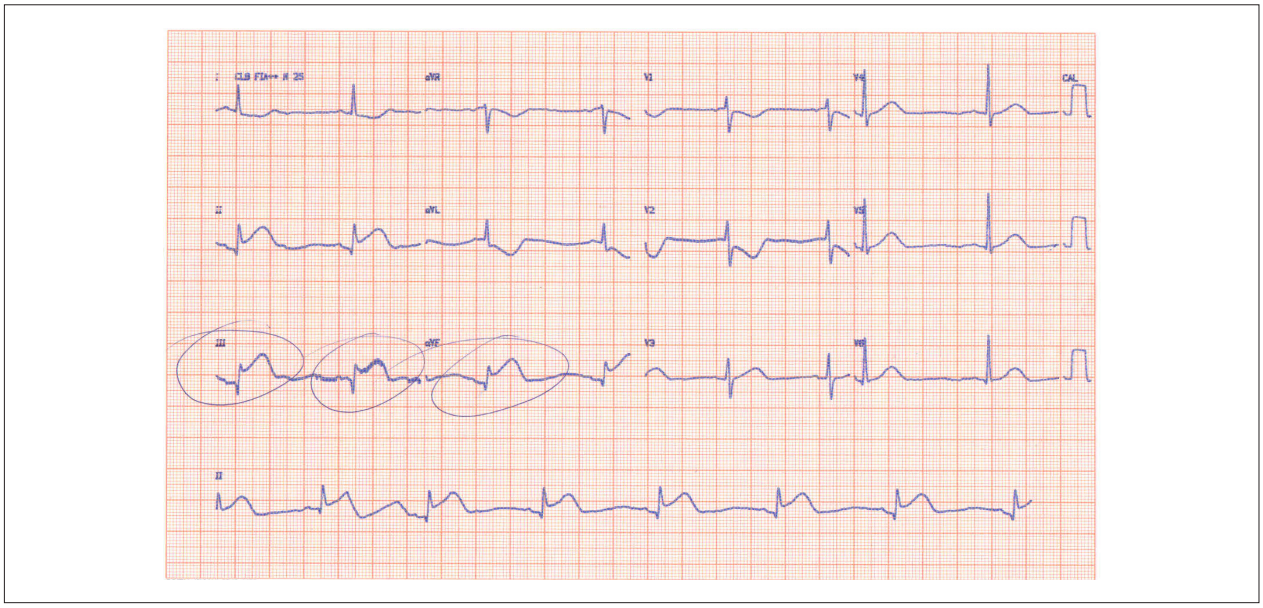

Figure 2 – *ECG: infarction in progress in the inferior wall, ST depression in V_1, V_2, and aVL.*

hypokinesia suggests that the patient may have experienced spontaneous reperfusion (which can occur in up to 33% of patients), with the right coronary artery reoccluding on the eighth day after infarction[3].

Inferior infarction can affect the right ventricle in 50% of cases; only 10%–15% of these cases include classic hemodynamic alterations such as hypotension, jugular distention, and pulmonary auscultation with no crackles, as in this case; although the sensitivity of this set of symptoms is less than 25%, it is very specific[4]. In general, right ventricular infarction occurs in patients with occlusion in the proximal third of the right coronary artery. This condition was found in this patient's coronary angiography, and the ECG revealed a new ST elevation in the inferior wall, a finding suggestive of reinfarction. However, she did not experience more precordial pain or increased troponin or CK-MB levels; these factors allow us to discard the hypothesis.

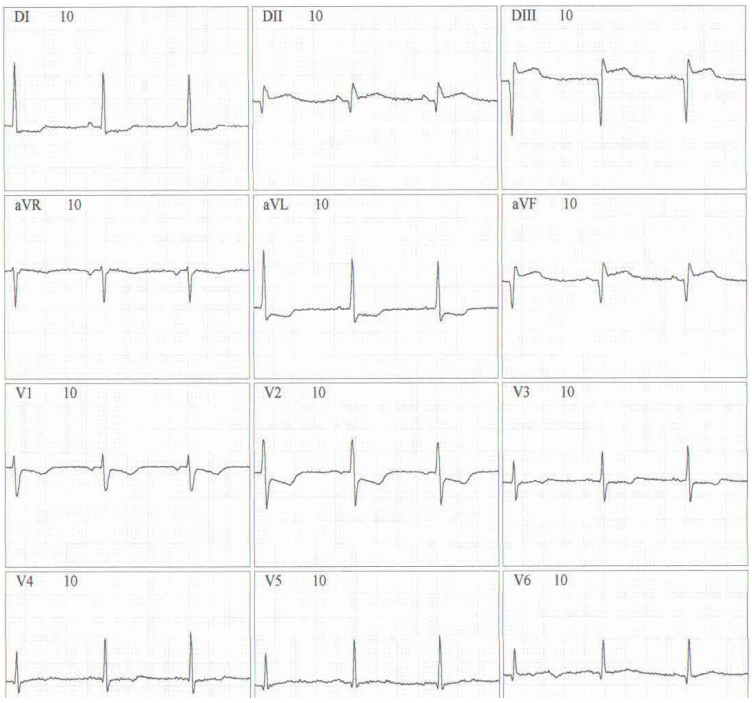

Figure 3 – *ECG: QRS complex parallel to the anatomic base-to-apex axis of the heart, QR waves (II, III, and aVF) with slight ST elevation and positive T waves, and ST depression in V_1, V_2, I, and aVL.*

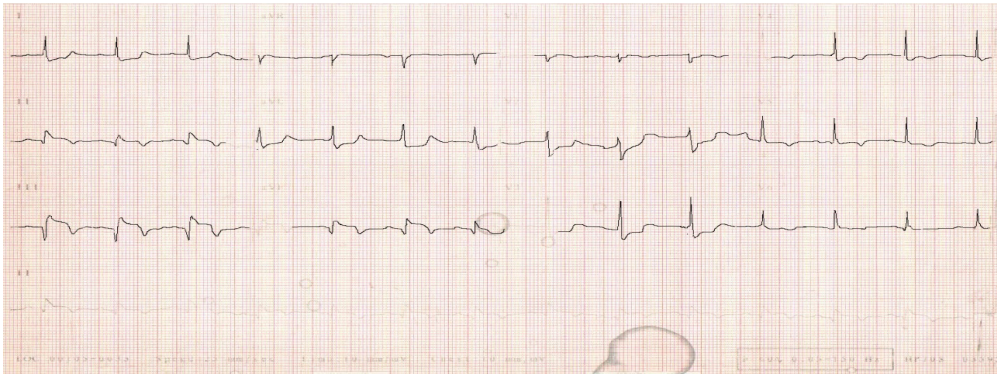

Figure 4 – *ECG: ST re-elevation in the inferior wall and repeated ST depression in lead I, aVL, V_1, and V_2.*

Therefore, the most probable hypothesis for the patient would be delayed post-infarction complications such as the rupture of the ventricular septum or left ventricular free wall. The rupture of the ventricular septum has an incidence of 0.2%–0.3%, and is most common between three and seven days after infarction[5,6]. Forty-seven percent patients may experience audible murmurs, which are more clearly heard at the lower left sternal border. Patients usually experience a worsened overall condition that can progress to congestive cardiac insufficiency[7,8]. The lack of both heart murmur and signs of pulmonary congestion make the hypothesis of interventricular septum rupture less likely. To confirm the diagnosis, another ECG would be needed for visualizing the shunt from the left ventricle right ventricle.

The incidence of ventricular free wall rupture ranges from 0.8% to 6.2% in cases of infarctions and is present in approximately 10% patients who die during the period of hospitalization for acute myocardial infarction. Most cases are associated with transmural infarction, and they are seven times more frequent in the left ventricle

than in the right ventricle. They are also more prevalent in anterior wall infarctions[8]. The rupture can be either complete or incomplete, a variation which results in different clinical presentations. Jugular distention, pulsus paradoxus, and cardiogenic shock may occur.

The rupture may be acute, subacute, or chronic. Acute ruptures are characterized by hemorrhagic pericardial effusion and result in high mortality rates. In case of subacute rupture, hemorrhagic pericardial effusion may be slow and repetitive, with the formation of thrombi between the epicardium and pericardial cavity, which limits hemorrhaging. Because this event occurs gradually, the patient may survive for hours or days, as seen in this case. Consistent or increased ST elevation in the leads can precede rupture, and an echocardiogram would confirm the location of the rupture or the presence of pericardial effusion, with or without tamponade[9]. In this case, the patient experienced new ST elevation in the inferior wall; however, there was not enough time to perform an ECG because of the quick progression of her case.

The patient underwent a coronary angiography and died after the procedure, which suggests the possibility of complications during the process. Coronary angiography is suggested in high-risk patients who present acute coronary syndrome without ST elevation or previous infarction, who have initially undergone thrombolysis treatment and experience post-infarction angina. Because this was a case of suspected reinfarction, a coronary angioplasty was performed and stents were implanted from the proximal portion to the distal portion, despite the fact that the right coronary artery was occluded in the coronary angiography. The most common complications of coronary angiography include cardiogenic shock, infarction, coronary artery dissection, stent thrombosis, stroke, the need for dialysis, and death; the latter occurs in 7.3% patients with EF $< 50\%$[10]. In this case, we considered it unlikely that the patient's death was caused by a complication in the procedure because her condition had already worsened before the procedure was performed.

We cannot neglect the possibility that death in this case was caused by pulmonary thromboembolism because patients who are hospitalized are often bedridden, which increases the risk of thrombosis. In patients with massive pulmonary thromboembolism, there may be an increase in pulmonary artery pressure, which leads to hypokinesia of the right ventricle, a decrease in cardiac deficit, and as a consequence, signs of hyperperfusion and hypotension, as seen in this case. Other findings and exams that can aid in the diagnosis include the presence of hemoptysis, cough, syncope, elevated D-dimer, and multi-detector tomography of the thorax to check for pulmonary emboli. The association between pulmonary thromboembolism and deep venous thrombosis occurs in 90% patients; however, in this case, no signs of thrombosis were present. Therefore, this diagnosis is less likely[11].

This patient experienced inferior wall infarction with ST elevation and after eight days displayed hypotension, jugular distention, and normal cardiac and pulmonary auscultation symptoms that then progressed to cardiogenic chock after coronary angiography. The patient did not respond to vasoactive drugs and died hours later. The principal diagnostic hypothesis is the rupture of the ventricular free wall with subacute progression, which is a delayed, post-infarction complication **(Dr. Bruno Aguiar Pinheiro, Dr. Alice Tatsuko Yamada).**

Diagnostic Hypotheses: Acute myocardial infarction with cardiogenic shock, based on mechanical complications such as rupture of the left ventricular free wall **(Dr. Bruno Aguiar Pinheiro, Dr. Alice Tatsuko Yamada).**

Autopsy

The heart weighed 540 g and was found to be slightly dilated in the left chambers. Transverse sections into the ventricles displayed a speckled myocardial appearance in the basal and mid segments of the inferior and septal walls (Figure 5). Complete rupture of the left posterior papillary muscle at its base was noted; the muscle itself was twisted because were the mitral valve cords in the corresponding commissure (Figure 6). Left atrial dilation was observed. The lungs were found to be heavier than normal (1050 g both combined), and were yellowish in color.

Histological examination of the heart revealed myocardial infarction approximately 1 week before death in addition to more recent subendocardial infarctions (within 1–2 days; Figure 7). After the right coronary stents were removed, the histological study revealed moderate diffuse atherosclerosis in addition to focal rupture and localized dissection of the posterior interventricular branch of the right coronary artery (Figures 8 and 9).

There were still signs of cardiogenic shock and acute pulmonary edema. The autopsy revealed bilateral hydronephrosis with signs of chronic pyelonephritis in addition to a simple hepatic subcapsular cyst, and the gallbladder was not present as a result of surgical removal **(Dr. Vera Demarchi Aiello).**

Pathological Diagnoses

Pathological diagnoses included systemic and coronary atherosclerosis; ischemic heart disease; acute inferoseptal myocardial infarction with left posterior papillary muscle rupture, acute mitral insufficiency, and bilateral chronic pyelonephritis.

Cause of death: Cardiogenic shock with bilateral acute pulmonary edema **(Dr. Vera Demarchi Aiello).**

Comments

Post-infarction myocardial rupture is a serious complication that most frequently occurs within the first week after infarction and mainly affects the free ventricular wall. It results in hemopericardium and death by cardiac tamponade. In most cases, it occurs in the transition between the necrotic and preserved myocardium. Intracardiac rupture can affect the ventricular septum, leading to interventricular communication or even, as in this case, papillary muscle rupture with acute mitral insufficiency.

Based on our experience at the Cardiology Institute, Hospital das Clínicas, School of Medicine of the Universidade de São Paulo (INCOR-USP), we have predominantly seen

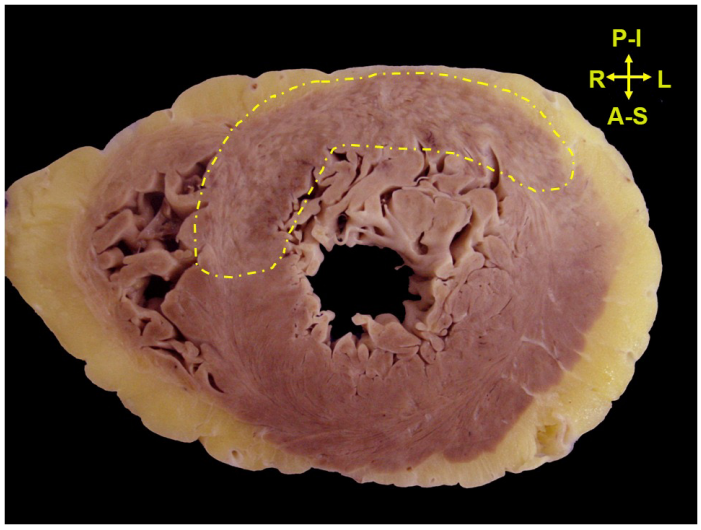

Figure 5 – *Transverse section into the ventricles showing a speckled myocardial appearance (area shown within the dotted yellow line). AS: anterosuperior; PI: posteroinferior; R: right; L: left.*

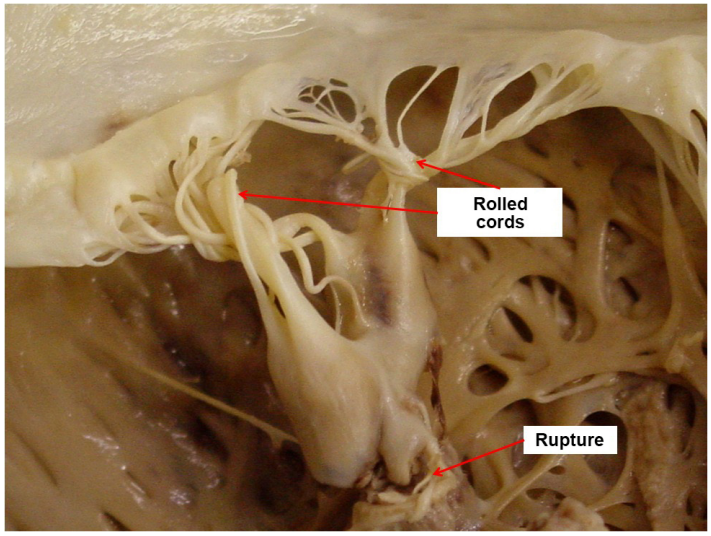

Figure 6 – *Detailed view of the ruptured left posterior papillary muscle, with twisting at its base and of the mitral valve cords in the corresponding commissure.*

the rupture of the free ventricular wall (approximately 80% cases) as well as ruptures of the septum and papillary muscle (16% cases)[12].

We have previously described post-infarction papillary muscle rupture as the cause of death in a case of dilated cardiomyopathy[13] and in a case of infarction resulting from septic emboli[14]. In the presence of generalized infection, the image of the ruptured muscle can be confused with vegetation in the ECG, and differential diagnosis with infective endocarditis becomes necessary.

The causes of cardiac rupture are not completely clear; however, some authors have reported a reduction in the αE-catenin protein in the myocardium of patients with this type of infarction. αE-catenin is a component of the intercellular adhesion complex of the myocardium[15].

Another study also revealed an increase in matrix metalloproteinases-8 and -9 in the ruptured muscle relative to the infarcted but not ruptured cardiac muscle[16] **(Dr. Vera Demarchi Aiello).**

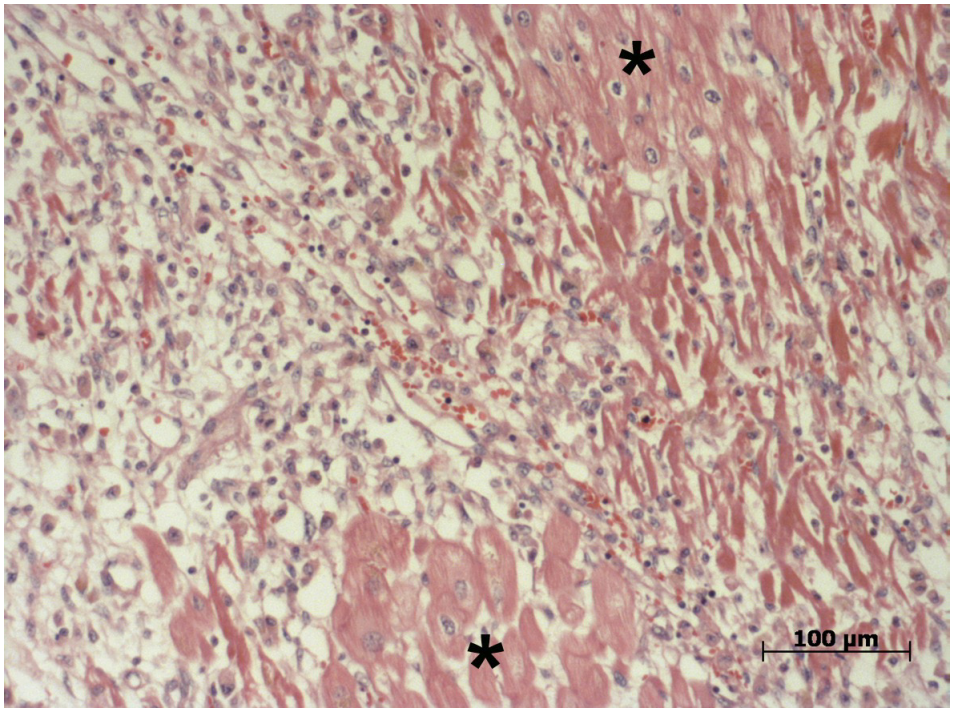

Figure 7 – *Micrograph of the periphery of the infarcted area showing the initial formation of granulation tissue with rare, newly-formed vessels and numerous histiocytes. The asterisks show preserved areas of the myocardium. H&E stain, 10× objective magnification.*

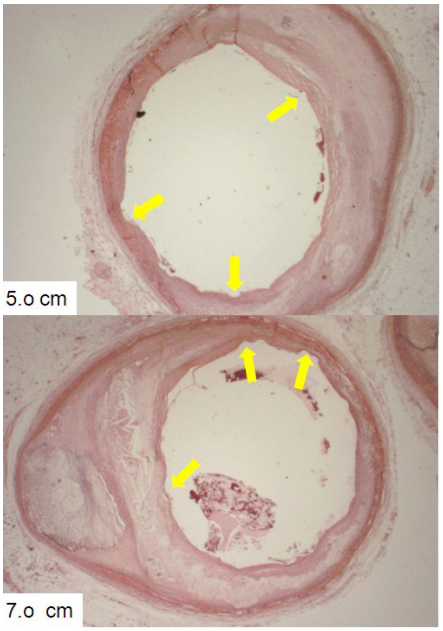

Figure 8 – *Micrographs of the right coronary artery at the fifth and seventh centimeters, showing eccentric atheroma plaques and calcification. Yellow arrows show points of compression by stent mesh (previously removed to allow histological sections). H&E stain, 1× objective magnification.*

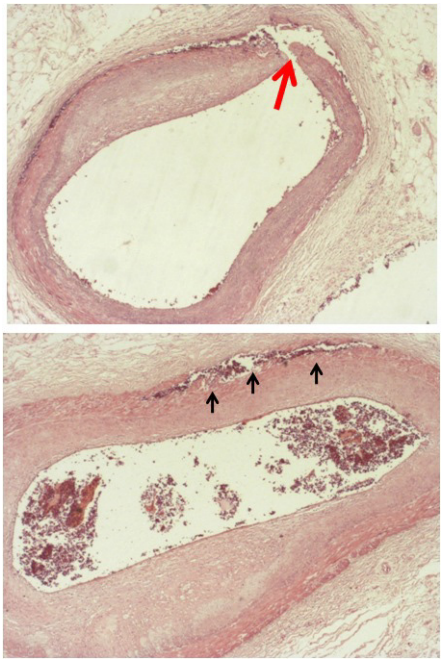

Figure 9 – *Micrograph(s) of the two distal segments of the posterior interventricular branch of the right coronary artery, showing rupture (red arrow) and dissection (black arrows) of the wall in addition to fibrous atheroma plaques. H&E stain, 2.5× objective magnification.*

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