

CASE REPORT: PATIENT WITH SEVERE THREE- VESSEL CORONARY ARTERY DISEASE

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Abstract: Introduction: Ischemic heart disease is caused by an imbalance in the blood supply and nutritional needs of cardiac tissue, often due to coronary stenosis of atherosclerotic origin, and is the leading cause of non-traumatic death in Brazil. **Methodology:** report and discussion of a case of a patient with severe coronary artery disease, of multivessel pattern, hospitalized for Acute Coronary Syndrome (ACS), addressing aspects related to the best management for diagnosis, risk stratification and treatment of the patient. **Results and discussions:** We reported the case of a patient with typical chest pain who sought care at a municipal emergency room, and an electrocardiogram was performed that showed ST-segment elevation in the wall. Routine medications for ACS were initiated, without improvement. The patient was referred to a tertiary hospital for invasive stratification (cardiac catheterization). The patient rapidly developed signs of cardiogenic shock. Urgent catheterization showed significant three-vessel coronary disease. Surgical revascularization was indicated after clinical stabilization. The patient is currently being followed up in an outpatient setting awaiting surgical scheduling. **Conclusion:** The case reported allows us to have a broad discussion regarding the various therapeutic options for the treatment of multivessel coronary artery disease: the classic option for myocardial revascularization surgery (the best therapeutic option for these patients, when possible); percutaneous intervention (either by isolated revascularization of the culprit vessel or by complete revascularization) – often chosen in the scenario of multivessel patients who present with ACS; and the option for clinical/drug treatment (especially useful for very elderly patients, with low life expectancy, with multiple comorbidities or with coronary arteries not suitable for receiving grafts or stents). In this complex clinical scenario,

the contemporary approach suggests that the choice must be individualized, taking into consideration, the clinical picture, age, comorbidities and coronary angiography (coronary anatomy) of the patient for wise decision-making that allows the patient a greater quality and quantity of life.

Keywords: Acute Myocardial Infarction; Heart Failure; Coronary Diseases

INTRODUCTION

Ischemic heart disease originates from an imbalance between the blood supply and the nutritional needs of the myocardium. In approximately 90% of cases in which the disease manifests itself acutely, there is an association between pre-existing atherosclerotic stenosis and current thrombosis and/or vasospasm of the coronary arteries. The narrowing of the arterial lumen can occur, together or in isolation, in any of the main arteries of the heart (left main coronary artery, left anterior descending artery, left circumflex artery and right coronary artery) or one of their branches. Acute myocardial infarction occurs when the imbalance is so intense (total and abrupt interruption of blood flow to that territory of the myocardium) that it causes necrosis of the heart muscle (VINAY KUMAR; ABBAS; ASTER, 2018). In Brazil, acute myocardial infarction (AMI) is the leading cause of death in the population (with an annual mortality rate of over 300,000 cases). The main risk factors for coronary heart disease include, among others: smoking, diabetes, dyslipidemia, systemic arterial hypertension, obesity, sedentary lifestyle, stress, male gender and family history of coronary artery disease (MOURA, 2024). In general, patients who have an AMI report intense and acute chest pain, described as a burning, tightness or precordial pressure, which radiates to the cervical, mandibular or left upper limb regions, of strong intensity, continuous, lasting more

than 10 minutes, which worsens with physical exercise and which may improve slightly with rest or the use of sublingual nitrate. In addition, some report nausea, vomiting, sweating, palpitations and dyspnea (JANOTTI, 2024). The diagnostic elucidation of AMI requires meticulous clinical and complementary resources, including: early performance of an electrocardiogram; detection of increased cardiac biomarkers in peripheral blood (such as troponin); and coronary morphological study through angiography (JANOTTI, 2024).

The Acute Myocardial Infarction, didactically, can also be classified as an Acute Coronary Syndrome (ACS). In general, we can classify ACS into two large distinct categories according to the electrocardiographic pattern: Acute Coronary Syndrome with ST-segment elevation (electrocardiographic correspondence of the end of depolarization and the beginning of repolarization of the ventricles) and Acute Coronary Syndrome without ST-segment elevation (BETT, 2024). This categorization is correlated with the magnitude of myocardial hypoflow during the coronary event, so that, in general, ST-segment elevation is the electrocardiographic product of intense and transmural ischemia of the heart muscle, resulting from a total or critical obstruction of one or more coronary arteries. Thus, it is possible to infer that acute myocardial infarction with ST-segment elevation is the most devastating acute manifestation of coronary artery disease, where there is a high probability of myocardial necrosis secondary to acute ischemic injury (OLIVEIRA, 2024). On the other hand, when ACS is not accompanied by ST-segment elevation, in general, there is no way to define that the ongoing myocardial ischemia will be sufficient to cause definitive damage to the myocardium (myocardial necrosis). Thus, during a non-ST-segment elevation ACS, myocardial ischemia may occur, which is generally not brutal or transmural

and may or may not result in necrosis of the myocardial tissue. Thus, after the first 24 hours, patients with non-ST-segment elevation ACS may or may not be reclassified to the diagnoses of non-ST-segment elevation AMI, Unstable Angina, or Chest Pain unrelated to myocardial ischemia.

Regarding therapy, early and adequate reperfusion of the affected tissue is mandatory, and may be performed through percutaneous coronary intervention or intravenous thrombolytic therapy. In addition, the prescription of antiplatelet agents, anticoagulants, beta-adrenergic blockers, and statins is recommended for therapeutic management, depending on the case in question (JANOTTI, 2024). In terms of complications, it is known that areas of electrical instability with potential foci of arrhythmias arise in regions of ischemia. Thus, although a massive infarction can cause a fatal cardiac mechanical disorder, approximately 80 to 90% of cases of sudden death after a coronary event (especially those that occur within the first 24 hours) are the result of ventricular fibrillation triggered by myocardial electrical instability (VINAY KUMAR; ABBAS; ASTER, 2018).

METHODOLOGY

In view of the above, this case report aims to describe the case of a patient who presented an acute myocardial infarction with ST-segment elevation and was diagnosed with significant stenosis of all 3 major arteries of the heart. To this end, the authors used information from the patient's medical records and images obtained during the hospital examination, and sought to preserve the identity of the reported subject. In addition, for theoretical improvement, a review of relevant studies in databases (PUBMED, SciELO and LILACS) and in the general medical literature was carried out.

RESULTS AND DISCUSSION

A 51-year-old male truck driver from the interior of São Paulo was previously healthy, sedentary, and a smoker with no history of cardiovascular disease or events. He denied alcoholism or illicit drug use, but reported smoking approximately 5 straw cigarettes per day for over 10 years. The patient initially sought care at an Integrated Care Unit (a Municipal Emergency Room for primary and secondary care) in a city in the Triângulo Mineiro region. After being diagnosed with AMI with ST elevation, the patient was referred to a Tertiary Hospital. Upon admission to tertiary care, the patient reported that he was in the city for work when, while having his first meal of the day, around 6 a.m., he noticed a sudden episode of tight chest pain, triggered at rest, of moderate intensity (scored as 6/10), radiating to the left upper limb, lasting 15 minutes and with spontaneous improvement, without association with other symptoms.

Due to the spontaneous improvement, the patient continued his scheduled activities for the day. However, around 10 a.m., he reported recurrence of chest pain, this time with worsening of intensity to very severe (9/10), associated with blurred vision and profuse sweating. The new episode of pain also started at rest and lasted for a long time, prompting him to seek a municipal emergency care unit. Upon arrival at the Municipal Emergency Room (secondary care), an electrocardiogram was performed which showed acute myocardial infarction with ST segment elevation in the AVR, aVF and DIII leads and ST segment depression in V2-V6 (Figure 1).

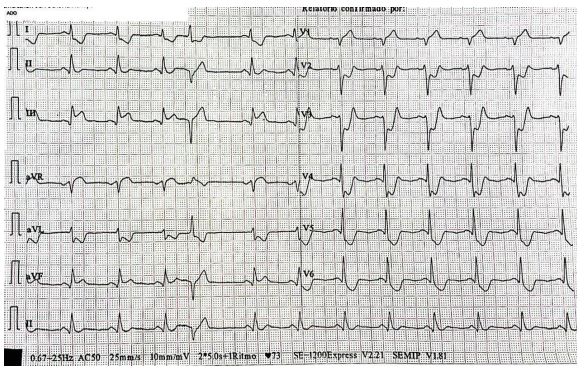


Figure 1: Electrocardiogram presented upon admission to the Municipal Emergency Room, identifying ST segment elevation in leads AVR, aVF, DIII and ST segment depression in V2-V6

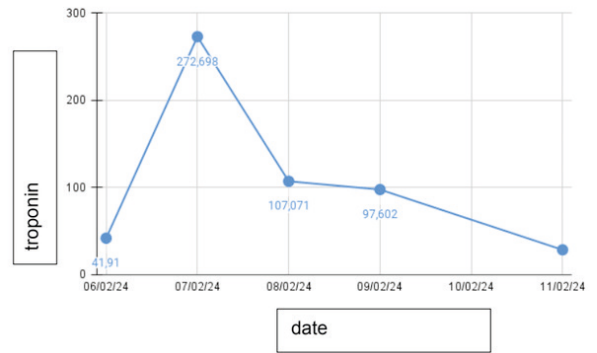


Figure 2: Troponin curve measured serially during hospitalization showing a higher peak 24 hours after the initial symptoms, with a gradual decrease in subsequent days

After identifying ACS with ST-segment elevation, the following were administered: isosorbide dinitrate (10 mg orally - PO), morphine (4 mg intravenously), aspirin (300 mg orally) and clopidogrel (300 mg orally) in a loading dose. However, given the worsening of the condition, with persistent chest pain, the patient was referred from a zero-bed hospital to a tertiary hospital in the Triângulo Mineiro region for invasive stratification, being admitted at approximately 1:00 p.m. (totaling a time interval of approximately 7 hours after the first episode and 3 hours after the recurrence of precordial pain - the most intense episode). The troponin level on admission to the University Hospital was 41.910 ng/mL (for a normal reference value < 0.01 ng/mL (Figure 2).

Upon admission, the patient was in good general condition, with normal color, hydrated, acyanotic, afebrile, and agitated due to intense pain. His body mass index (BMI) was 26.31 kg/m². On evaluation of the cardiovascular system, cardiac auscultation revealed rhythmic, normal-sounding sounds in two beats, without murmurs, with capillary refill time of less than 2 seconds, blood pressure of 112 x 80 mmHg, heart rate of 86 bpm, symmetrical radial pulses with resolved amplitude, hemodynamically stable, but tending toward hypotension. Regarding the respiratory system, he presented physiological vesicular murmur, without adventitious sounds, without respiratory distress, and with oxygen saturation of 98% on room air. On abdominal physical examination, the abdomen was flat, flaccid, with bowel sounds present, painless on palpation, and without masses or visceromegaly. He remained oriented and conscious in time and space during admission, with a report of partial improvement in the precordial pain after initial measures (including morphine) that had been administered.

The diagnosis of acute myocardial infarction with ST-segment elevation in the inferior wall was made, initially classified as Killip 1, due to the fact that the patient had no

evident clinical signs of heart failure. However, during the first hours of care at the Tertiary Hospital, the patient developed a decrease in blood pressure levels (arterial hypotension), and support with vasoactive drugs was offered due to probable cardiogenic shock. He was referred to the hemodynamic unit for urgent cardiac catheterization.

Catheterization and coronary angiography were performed by puncturing the right radial artery after infusion of heparin 5000 U, monocordil 10 mg (intra-arterial). The following was observed: trifurcated left coronary trunk, with an eccentric obstructive lesion 30-40% distal; well-developed anterior descending artery, with total occlusion (100%), with a chronic appearance, in the middle segment of the vessel; circumflex artery classified as moderately developed, with total occlusion (100%), also probably chronic, in the middle segment; well-developed, dominant right coronary artery, with total occlusion (100%) associated with images suggestive of thrombi (probably acute) in the proximal segment of the vessel (Figure 3).

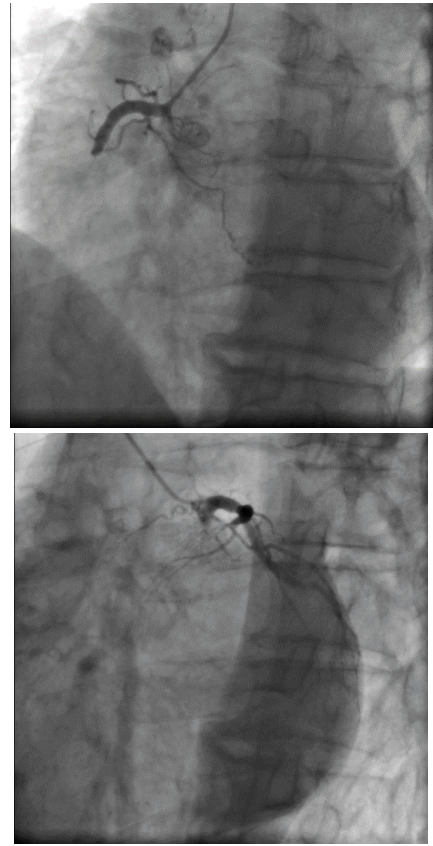


Figure 3: Right and left coronary angiograms, prior to angioplasty, showing total occlusion of arteries: anterior descending, circumflex and right coronary.

At the time, right coronary angioplasty was performed with implantation of a 3.5x30 mm drug-eluting stent, involving the proximal and middle segments of the vessel. A control coronary angiography was performed, which identified a good angiographic result, with no signs of dissection or thrombus, no significant residual lesions, and distal flow to the right coronary artery TIMI III and BLUSH III. The emergence of important collaterals refluxing to the left coronary artery was also identified, with no signs of complications (Figure 4).

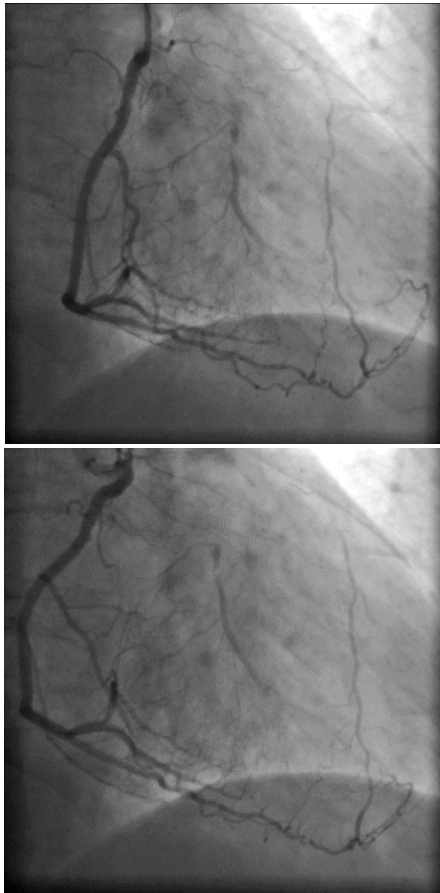


Figure 4: Catheterization, after angioplasty in the right coronary artery, with implantation of 1 drug-eluting stent, with improvement in coronary perfusion (TIMI III, Blush III) and identification of good collateral flow to the Anterior Descending and Circumflex arteries.

During angioplasty, the patient developed hemodynamic instability, presenting with hypotension, complaints of dyspnea, bradycardia and heart rhythm oscillations with atrial fibrillation pattern (classified as Killip IV). After the procedure, a new electrocardiogram was performed which showed improvement in reperfusion with reduction in ST-segment elevation (Figure 5).

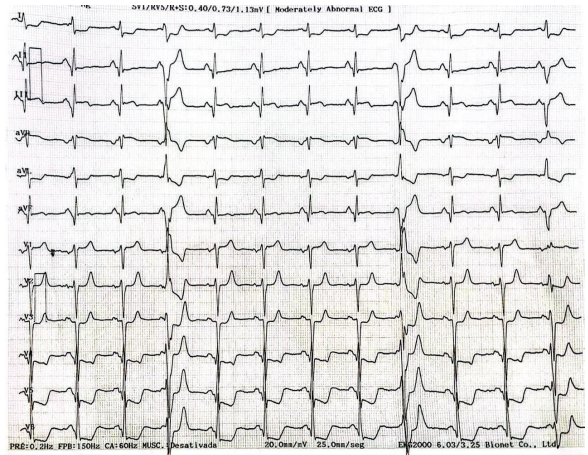


Figure 5: Electrocardiogram performed after angioplasty showing Sinus Rhythm, Ventricular Extrasystoles, Inferior Inactive Electrical Zone, improvement in ST elevation in aVF, aVR and DIII, but maintenance of ST depression in the anterior wall.

Transthoracic echocardiogram identified significant left ventricular (LV) systolic dysfunction due to segmental alteration in contractility, LV ejection fraction of 29%, left atrium of 36 mm, in addition to significant concentric left ventricular hypertrophy, and grade 1 left ventricular diastolic dysfunction. Chest X-ray showed the presence of pulmonary congestion (Figure 6).

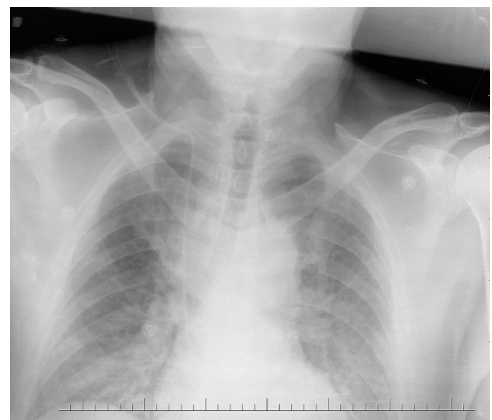


Figure 6: Chest X-ray (of fair technical quality), allowing identification of significant pulmonary congestion, especially perihilar, cephalization of the vascular network, with diffuse bilateral interstitial infiltrate, more predominant in the lung bases, performed on the day of admission.

The case was discussed jointly with the cardiac surgery and cardiology teams, and a decision was made to perform myocardial revascularization surgery within 60 days. The decision in favor of performing myocardial revascularization surgery (but only after a few weeks) was justified by the context of a recent, severe ischemic event (with stunning of a large part of the myocardium) and by the fear of prematurely discontinuing dual antiplatelet therapy due to the important contribution of the right coronary artery in the emission of important collaterals to the left coronary artery (high risk of stent thrombosis in case of premature discontinuation of antiplatelet agents). The patient progressed well, with progressive hemodynamic improvement, and was discharged for follow-up in the cardiac surgery outpatient clinic, after being discharged from the hospital, which occurred after 6 days of hospitalization in the Chest Pain Unit (TPU). The patient returned to his hometown hemodynamically stable, eupneic, saturating well in room air, and with no complaints when walking through the corridors of the Hospital Ward. Advised to use continuous medications: antiplatelet agents (Aspirin and Clopidogrel), beta-blockers (Metoprolol), statins (Atorvastatin), antiarrhythmics (Amiodarone due to intermittent atrial fibrillation during hospitalization), angiotensin-converting enzyme inhibitors, spironolactone (due to ventricular dysfunction) and proton pump inhibitors (PPIs).

FINAL CONSIDERATIONS

Based on the case described, the medical team's decision to indicate coronary artery bypass grafting (CABG) after the initial percutaneous coronary intervention (PCI) is related to the fact that the patient had multivessel coronary artery disease with two chronic occlusions (of the circumflex and anterior descending arteries). The approach to chronic occlusions has a lower success rate and long-term stent patency. In addition, the reduction in left ventricular ejection fraction and a large area of potentially ischemic myocardium also favor the choice of CABG to obtain complete, more effective and safer myocardial revascularization. However, patients with multivessel coronary disease, in high-risk situations, can also undergo multivessel PCI (in two or more vessels) that can be performed simultaneously with percutaneous recanalization of the vessel responsible for the AMI or at separate times (staged percutaneous revascularization). The staged approach can also be programmed, ideally, during the same hospitalization or at another time after a period of outpatient observation. (SERRUYS et al., 2009)

Thus, the choice between maintaining only drug (clinical) treatment or indicating a surgical (CABG) or percutaneous (PCI) revascularization procedure in patients with multivessel disease (in this case, three-vessel disease) is influenced by a series of factors, which include clinical aspects (age, comorbidities and life expectancy of the patient); angiographic aspects (which include several aspects such as: the number of vessels involved, their caliber, the degree of calcification and associated thrombi and their tortuosity, the amount of myocardium supplied by the affected vessels, the anatomical complexity of the lesions requiring revascularization, the probability of obtaining complete revascularization; and other aspects such as the patient's and family's

preference regarding the various therapeutic proposals that can be discussed and offered by the heart team (cardiologists, hemodynamicists and cardiac surgeons). In view of this, at the time of decision-making, the patient must be informed about the relative risks of death, stroke, worsening of renal function, infection, bleeding, need for new revascularization procedures and chances of improving quality of life and increasing life expectancy for each of the selected strategies. (SERRUYS et al., 2009). In the reported case, the option for CABG was essentially due to the need to obtain complete myocardial revascularization and the probable technical difficulty of Angioplasty in obtaining the recanalization of chronic occlusions of chronically compromised arteries (Anterior Descending and Circumflex).

Therefore, although there are several national and international guidelines that direct decision-making regarding the therapeutic

approach in terms of revascularization to be proposed (whether percutaneous or surgical), the final decision on the therapy to be offered to the patient and family must take into account all of these individual factors (clinical, angiographic and preferred aspects) of the patient and must always be individualized and shared among all members of the heart team, attending physician, patient and family. After all, all therapeutic options offer their potential benefits and risks that are, in most cases, impossible to estimate accurately for each individual. Therefore, the ideal therapy for this patient with multivessel coronary disease with left ventricular dysfunction is a controversial decision, which, although more randomized trials are still needed in the medical literature for definitive general guidance, will continue to be very individualized and variable according to each patient.

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