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CHEST PAIN IN THE EMERGENCY ROOM: IDENTIFICATION, CLASSIFICATION AND MANAGEMENT OF ACUTE CORONARY SYNDROME

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Geovanna Araújo Maciel[1], Débora Priscilla Araújo Maciel[2], Marcia Caparroz Nogueira[3], Eloisa Gomes Sartin[4], Lilian Gomes Barbosa[5], Janaína Gonçalves de Souza[6], Letícia Almeida Quixabeira[7], Ueverton Rodrigues de Sousa[8], Camila Gigante Rocha[9], Daiane Furtado Maluf[10], Vinicius Veidrick Melo[11], Adrian Vinícius de Carvalho Dantas[12], Poliana Rafaela dos Santos Araújo[13]

[1] Medical Student
Afya – Faculties of Medical Sciences - Itabuna campus
LATTES: https://lattes.cnpq.br/9621422481395770
E-mail: araujoo.geovanna@gmail.com
[2] Medical Student
Federal University of Acre
LATTES: http://lattes.cnpq.br/0785959970453825
E-mail: deborapriscilla.ufac@gmail.com
[3] Medical Student
Universidade Brasil - campus Fernandópolis – São Paulo
LATTES: http://lattes.cnpq.br/8527351317611129
E-mail: macaparroz@gmail.com
[4] Graduating in Medicine
Universidade Brasil - campus Fernandópolis – São Paulo
LATTES: http://lattes.cnpq.br/5426588763687359
E-mail: eloisag.sartin@gmail.com
[5] Medical Student
Universidade Brasil - campus Fernandópolis – São Paulo
E-mail: gomeslilian82@yahoo.com.br
[6] Medical Student
Universidade Brasil - campus Fernandópolis – São Paulo
E-mail: janag.souza@hotmail.com
[7] Medical Student
UNIRV - University of Rio Verde – Goiás
E-mail: leticia.quixabeira@academico.unirv.edu.br
[8] Graduating in Medicine
Universidade Brasil - campus Fernandópolis – São Paulo
LATTES: http://lattes.cnpq.br/6072516728927455
E-mail: prof.pinhate@gmail.com
[9] Medical Student
Afya – Faculties of Medical Sciences - Itabuna campus
LATTES: http://lattes.cnpq.br/0351017908375162
E-mail: giganterochac@gmail.com
[10] Medical Student
Afya – Faculties of Medical Sciences - Itabuna campus
E-mail: daimaluf13@gmail.com
[11] Graduating in Medicine
Afya – Faculties of Medical Sciences - Itabuna campus
E-mail: vinicius.veidrick@hotmail.com
[12] Graduating in Medicine
Uninorte University Center
LATTES: http://lattes.cnpq.br/7470290760863272
E-mail: adrianviniciuscd@gmail.com
[13] Medical Student
Federal University of Acre

and Gabriela Pederiva Engler[14]

ABSTRACT
Chest pain is one of the most common complaints in emergency departments, and can be classified as cardiac and non-cardiac in origin. When of cardiac origin, it can indicate highly serious and life-threatening conditions such as Acute Coronary Syndrome (ACS) and pulmonary thromboembolism. Cardiovascular diseases are the leading cause of death in the world and represent the leading cause of death in Brazil since 1960. According to the World Health Organization (WHO), in 2019, 18 million deaths were due to cardiovascular diseases in the world. Among the main causes of death from cardiovascular diseases, ischemic heart disease stands out, which represents the cause of 9 million deaths in the same year. According to DATASUS, in 2021 in Brazil, approximately 115,000 deaths from ischemic heart disease were described, reflecting the global scenario. Its etiology is multifactorial, but it is worth noting that the disease is closely related to preventive risk factors, such as smoking, obesity, poor diet, sedentary lifestyle, and poor control of underlying diseases such as hypertension and diabetes. The result of these poor lifestyle habits is high morbidity and mortality and increasing socioeconomic impact since the management of this comorbidity represents 8.3% of all hospitalizations and 18% of SUS costs according to DATASUS in 2022. Given this scenario, it is essential to implement strategies aimed at changing lifestyle habits, in addition to emphasizing early diagnosis, appropriate classification, and efficient management of patients, to reduce morbidity and mortality associated with cardiovascular diseases.

Keywords: Chest pain. Urgency. Acute Coronary Syndrome. Classification. Handling.

LATTES: http://lattes.cnpq.br/6900249896550982
E-mail: poliana.rsa@gmail.com
[14] Medical Student
Federal University of Acre
LATTES: http://lattes.cnpq.br/8928117307993553
E-mail: gabrielap1010@hotmail.com

INTRODUCTION

Acute Coronary Syndrome (ACS) is a set of signs and symptoms caused by myocardial injury caused by the sudden reduction or blockage of blood flow in the coronary arteries, leading to myocardial ischemia. It is estimated that ACS is responsible for 20% of deaths in the world. It occurs in Brazil from 300 to 400 thousand cases/year. Although we have evolved in the use of drugs to prevent these events such as ASA and statins, we still have a high incidence (DE SOUZA et al., 2021; DA SILVA et al., 2023).

This syndrome encompasses three main clinical presentations, unstable angina, non-ST-elevation myocardial infarction NSTEMI, and STEMI-elevation myocardial infarction (STEMI). They are one of the 3 main classifications of ACS responsible for angina of strong intensity in the emergency context (FERNANDES et al., 2020).

ACS represents the clinical expression of the destabilization of a coronary atherosclerotic plaque, after suffering a rupture or erosion and initiating the healing cascade of this plaque, thus causing the thrombus on it, from platelet aggregation and activation of the coagulation cascade. This plaque, together with the thrombus, can partially or completely occlude the lumen of the vessel, which limits the supply of oxygen to the myocardial tissue and, thus, determines the clinical syndrome (SANTOS et al., 2023).

The clinical condition of patients with ACS is caused precisely by the lack of O2 in the cardiomyocytes, which are the heart cells. Through the mechanism of ischemia, the death of these cells results. For this reason, there is a need for early diagnosis and intervention of this patient. The damage caused by the death of cardiomyocytes is irreversible, so the sooner the blood flow to the coronary arteries is restored, the more heart muscle will be spared (ASUNÇÃO et al., 2020).

The main clinical findings found in a patient with ACS are characterized by precordial or retrosternal pain of strong intensity, of an oppressive nature, lasting for a long time (>20 minutes), which may radiate to MSE, dorsum, or mandible, which increases its intensity with stress or exertion, and may often be accompanied by nausea, vomiting, dyspnea, epigastric pain, and diaphoresis. There is a classic clinical semiological sign for angina and chest pain in ACS, called Levine's sign, which is an important semiological finding. This sign is manifested when the patient places a clenched fist on the chest when complaining of chest pain (PAIVA et al., 2021).

There are some atypical symptoms in ACS, where the patient will not experience typical chest pain. These symptoms may be present in women, diabetics, patients with CKD, or dementia. These are symptoms known as ischemic anginal equivalents that can manifest as: sweating, pallor, dyspnea, dyspepsia, nausea, vomiting, and palpitations (PAIVA et al., 202; SANTANA et al., 2021).

The signs and symptoms of these patients vary according to the intensity due to some factors, such as the extent of heart muscle perfused by the obstructed artery, severity and duration of myocardial ischemia, and the degree of obstruction of the vessel lumen (ASUNÇÃO et al., 2020).

The determination of the 3 main types of ACS is based on the performance of some complementary exams, the first of which is the electrocardiogram. If there is an ACS, it is possible to identify whether or not it has ST-segment elevation. If there is ST-segment elevation, I classify it as ST-segment elevation myocardial infarction. If ST-segment elevation is not evidenced, I can have two types of ACS, non-ST-segment elevation myocardial infarction (NSTEMI) or unstable angina. What differentiates the two types is the elevation of biomarkers, the so-called tumor necrosis markers, which will increase in infarction and not in angina (FERNANDES et al., 2020).

ACS is a medical emergency and requires rapid diagnosis and prompt treatment to reduce morbidity and mortality. Management includes the use of antiplatelet agents, anticoagulants, vasodilators, beta-blockers, and, in some cases, percutaneous coronary intervention or coronary artery bypass grafting surgery (PAIVA et al., 2022).

OBJECTIVE

The general objective of this study is to identify, classify, and determine the management of Acute Coronary Syndrome, addressing its main clinical manifestations, necessary complementary tests, conducts used, and strategies to maximize the safety and efficacy of the established therapy. The aim is to provide a comprehensive and practical view for application in the clinical context.

The specific objectives

1. Identify the main risk factors for cardiovascular disease
2. Determine the diagnosis of Acute Coronary Syndrome
3. Name the main classifications of Acute Coronary Syndrome
4. Establish the appropriate management of Acute Coronary Syndrome

5. Emphasize the importance of prevention, early diagnosis, and proper management

METHODOLOGY

Considering that theoretical studies are an indispensable basis for field and laboratory research, we opted for conceptual deepening and searching for official data on the object of study, allowing the knowledge of reality as well as the possibility of critical reflection on the subject within the scope of Brazilian reality.

Based on the understanding of Creswell (2007) for whom the Literature Review is configured as a preliminary stage of scientific studies, then the research is a Literature Review in which articles published in the National Library of Medicine (Pubmed), Virtual Health Library (VHL), Web of Science, Lilacs and Capes Journals were used as the basis of the study by descriptors obtained by the Health Sciences Descriptors (DeCS) of the VHL.

This is a literature review of articles published in the National Library of Medicine (Pubmed), Virtual Health Library (VHL), Web of Science, Lilacs and Capes Journals by descriptors obtained by the Health Sciences Descriptors (DeCS) of the VHL. The following descriptors were searched: Chest pain AND urgency AND acute coronary syndrome AND management in "All fields".

For the selection of articles, the following steps were followed: (I) search for articles in the databases; (II) reading of titles and abstracts, with analysis according to the eligibility criteria and; (III) full-text analysis of the papers, including in the systematic review only those required by the inclusion criteria and did not meet any of the exclusion criteria.

As inclusion criteria, published studies were selected if they met the following criteria: (1) studies involving Acute Coronary Syndrome; (2) studies that had as an object of study the indications, classifications, strategies, and management of patients diagnosed with Acute Coronary Syndrome; (3) articles published in the last 4 years. There were no restrictions on sample size or foreign language.

As exclusion criteria, articles were excluded that: (1) were published before 2020; (2) studied situations that do not include the management of Acute Coronary Syndrome; (3) were duplicates; (4) they were not directly related to Acute Coronary Syndrome, its indications, techniques, strategies and challenges;

RESULTS AND DISCUSSION

Acute Coronary Syndrome is a myocardial injury due to acute ischemia. It represents the leading cause of death in the world. Despite the advancement of studies and medications, the overall mortality of ACS remains high, as most patients, about 50%, die within the first hour of the event, usually even before medical support (PAIVA et al., 2022).

Understanding the pathophysiological mechanisms of ACS is essential to understanding its clinical manifestations and management. Its pathophysiology is based on the atherosclerotic plaque, which is formed by an accumulation of LDL and cholesterol in the subendothelium, and at a certain time this plaque can rupture or erode caused by some hypertensive peak or a fragility in the fibrotic layer of the plaque itself. This rupture leads to the exposure of the lipid content to the light of the vessel, thus the formation of the white thrombus, which is formed by platelets, begins. These send biochemical signals for the subsequent formation of a red thrombus, composed of a fibrin network that "traps" red blood cells. The result of the formation of the red thrombus is total or partial obstruction of a coronary vessel. Where it rapidly reduces the flow of O2 in the muscle irrigated by the affected vessel. Sometimes this thrombus can move to another vessel and cause total occlusion and ischemia of a smaller vessel (BODANESE et al., 2020; JOHNSON et al., 2021).

Since ACS is defined as a myocardial injury due to acute ischemia, there are other causes in addition to atherosclerotic plaque that cause ACS. As occurs in coronary vasospasm or Prinzmetal's Angina induced by some medication or cold. Another cause is coronary artery dissection, which can take blood to other layers of the vessel and cause occlusion of the vessel. As well as systemic or gaseous embolisms, septic embolisms due to endocarditis, atrial fibrillation, ventricular or atrial thrombus, and myxoma. The imbalance of supply x demand, coronary vasculitis such as Takayasu, or Kawasaki, or iatrogenic through catheterization or angioplasty (ZIPES et al., 2022).

Understanding the pathophysiology of acute coronary events is essential to treatment. Understanding that most cases of ACS are of atherosclerotic etiology and knowing the pathophysiology of the evolution of the coronary event, it is understandable why the management of these patients is done with antiplatelet agents and anticoagulation. However, if there is another non-atherosclerotic condition for ACS,

treatment should be individualized. For example, a patient with septic coronary embolism secondary to endocarditis should have the underlying infectious cause as a priority treatment, just as the patient with coronary vasospasm has the use of coronary vasodilators as a key element in the treatment (SANTOS et al., 2023).

The evaluation of patients with suspected ACS in the emergency room should be performed initially through physical examination and clinical history directed to the focus on chest discomfort, signs and symptoms of acute heart failure secondary to ACS, the patient's cardiac history, the identification of risk factors for ACS, and historical characteristics that may preclude the use of fibrinolytic. Perform the electrocardiogram and interpret in the first 10 minutes, the dosage, if necessary, of the levels of cardiac biomarkers (DO NASCIMENTO et al., 2021).

The main clinical manifestations expected are typical angina, determined by the presence of all three of the following characteristics, substernal chest pain or discomfort, caused by exertion or emotional stress, relieved by rest or nitrates within a few minutes. Atypical angina, on the other hand, meets two of these characteristics mentioned above, and non-anginal chest pain meets only one or none of the characteristics (PAIVA et al., 2021).

There are three main clinical presentations of unstable angina, angina crescendo is the previous angina that is more frequent, with episodes of longer duration or with a lower threshold. Recent-onset angina with onset less than 2 months ago and intensity CCS III or IV. Angina at rest, occurs when the patient is at rest, lasting more than 20 minutes, occurring for a maximum of 1 week (PAIVA et al., 2021).

It is also important to highlight the anginal equivalents that are represented by dyspnea caused by systolic or diastolic dysfunction causing pulmonary congestion, fatigue and fainting sensation caused by low cardiac output, palpitations caused by tachyarrhythmias secondary to ischemia, nausea, vomiting, sweating, and pallor, which are neuro vegetative symptoms. These anginal equivalents are mainly present in diabetic patients, women, patients with CKD, or dementia (JÉSSICA et al., 2020; SANTANA et al., 2021).

Physical examination in ACS usually has few clinical findings, but they are classic for early identification of the situation. The physical examination should be rapid, objective, and directed to look for the main findings suggestive of complications and to exclude probable other diagnoses, such as pulmonary congestion, looking for

progressive crackles, cough with foamy and pink secretion thinking about acute pulmonary edema, and looking for B4 thinking about diastolic dysfunction. It is important to seek tissue hypoperfusion, cardiogenic shock with hypotension and tachycardia, B3 thinking about systolic dysfunction, lowered level of consciousness, and delayed capillary refill with cold extremities. It is also possible to identify some probable complications on physical examination, such as murmurs of acute mitral regurgitation and VSD. And exclude aortic dissection, due to the difference in pulses and pneumothorax seeking auscultation abolished in one of the hemithorax and pericarditis due to pericardial friction (PAIVA et al., 2021; DE OLIVEIRA et al., 2024).

Therefore, the main methods of identifying ACS include clinical evaluation, electrocardiogram (ECG) within 10 minutes of patient arrival, and cardiac biomarkers, such as ultrasensitive troponin, conventional troponin, CK-MB mass, and CK-MB activity. The best of them is ultrasensitive troponin. It is important to highlight that even though biomarkers are an important test for the diagnosis of ACS, it is not a specific test for ACS since they are markers of myocardial injury, which can be present in other pathologies such as heart failure, chronic kidney disease because it is not possible to excrete them properly, in myocarditis, pericarditis, transplant rejection, chemotherapy, direct or indirect cardiac trauma (ABRANTES et al., 2024).

The diagnostic flow is represented by the ECG within 10 minutes after arrival at the hospital in every patient with clinical suspicion, by anamnesis and physical examination of ACS. Of the ECGs performed in the emergency room, 50% are normal, even without electrocardiographic changes, If the patient persists with classic symptoms of ACS, the electrocardiogram should be serial. In the situation of ECG abnormalities, we mainly have NSTEMI and STEMI (main alterations are ST infra or ST-wave or ST-wave alterations) (PAIVA et al., 2022; MATOS et al., 2022).

Non-ST-elevation myocardial infarction (non-ST-elevation AMI) is characterized by myocardial necrosis evidenced by biomarkers, without ST-segment elevation on the electrocardiogram. On the other hand, ST-segment elevation myocardial infarction (STEMI) occurs when there is complete obstruction of the coronary artery, causing extensive myocardial necrosis and ST-segment elevation on the electrocardiogram. Unlike unstable angina, which occurs when there is an insufficient blood supply to the heart, without significant damage to the heart muscle (FURLANETTO et al., 2023).

The Killip and Kimball classification is necessary to assess the prognosis of AMI based on signs and symptoms of left ventricular failure. The higher the Killip, the more severe the SCA. Patients with Killip I have no signs of HF and the risk of obtaining it is 2-3%, those with Killip II have signs of mild HF, with rales in the bases and B3, and the risk of death is 8-10%, Killip III patients already have acute pulmonary edema and their risk of death is 20-25%, patients classified as Killip IV are already in cardiogenic shock and their risk of death is 45-70% (CEDRO et al., 2021).

After the diagnosis of ACS, risk stratification is necessary, through the TIMI (Thrombolysis in Myocardial Infarction) risk score, and the GRACE (Global Registry of Acute Coronary Events) is an index that evaluates the risk of death of patients with acute myocardial infarction (AMI) (CEDRO et al., 2021; CONTATO et al., 2023).

The initial management of these patients includes the administration of oxygen, antiplatelets, anticoagulants, nitrates, heparins, statins, and ACE inhibitors, among other medications, in selected cases, percutaneous coronary intervention (PCI) or coronary artery bypass grafting surgery (FERNANDEZ et al., 2024; NUNES et al., 2020).

Oxygen is a necessary therapy for patients with ACS with pulmonary congestion or SatO2 below 90%. In other situations, its use is discouraged. To avoid hyperoxia and iatrogenies. Dual antiplatelet therapy: acetylsalicylic acid and another antiplatelet agent, such as clopidogrel and ticagrelor, is required in the form of a loading dose (DE CASTRO et al., 2024).

The use of acetylsalicylic acid is mandatory, at a dose of 160 to 325 mg, because it reduces mortality and has a rapid antiplatelet effect by blocking the synthesis of thromboxane A2, interfering with the metabolism of arachidonic acid and inhibiting the formation of cyclooxygenase 1, a fundamental enzyme in the platelet aggregation process. Because it irreversibly inhibits COX-1, its effect persists as long as platelet survival lasts from 7 to 10 days. And its use after an AMI is for life in a maintenance dose, to avoid new AMI conditions. In case of allergy to acetylsalicylic acid, antiplatelet therapy should be started only with clopidogrel (CONSERVA et al., 2024; SOARES et al., 2023).

The use of clopidogrel at a loading dose of 300 mg is extremely necessary along with the use of ASA. Its maintenance dose is 75 mg/d. It is important to note that in patients over 75 years of age, the loading dose should be 75 mg/d. Its use is indicated

for all patients with UA or NSTEMI in whom clinical treatment or early approach to coronary lesion with angioplasty is planned. Its use should be suspended 5 to 7 days before the surgical approach for coronary artery bypass grafting, due to the high risk of severe bleeding in the postoperative period (DARZÉ et al., 2024).

Ticagrelor or prasugrel represents a new group of antiplatelet agents and can be used to replace clopidogrel for cases of UA/NSTEMI in which catheterization is used early in the acute setting of coronary insufficiency. After stenting, one of these drugs must be maintained for up to one year after the procedure (DE CASTRO et al., 2024).

The use of nitrates helps relieve pain as it improves congestion and hypertension. They should not be administered before the ECG, as their use is contraindicated in right ventricular AMI, which is present in 30% of cases of lower wall AMI. Its use should be avoided in hypotension, bradycardia below 60 bpm, or in cases of use of phosphodiesterase inhibitors for erectile dysfunction for less than 24 hours sildenafil and tadalafil for 48 hours (DARZÉ et al., 2024).

The use of morphine is indicated for pain reduction, this occurs because it promotes congestion improvement. It is indicated when pain is persistent or refractory even after nitrate. Do not use in case of SBP less than 90 mmHg (SOUZA et al., 2021).

Glycoprotein IIb/IIIa inhibitors are part of this class: abciximab, eptifibatide, and tirofiban. One of them should be used in conjunction with acetylsalicylic acid, clopidogrel, and heparin for all patients with high-risk UA or NSTEMI, associated with early approach with angioplasty. When this is not possible, tirofiban and eptifibatide are the drugs of choice. Abciximab can only be offered if the early approach with angioplasty is effectively performed (PAIVA et al., 2022).

The use of beta-blockers is initially recommended with metoprolol, it is indicated in moderate and high-risk angina and the presence of ischemic chest pain but contraindicated in patients with a history of bronchospasm, hypotension, moderate to severe left ventricular dysfunction, PR interval greater than 0.24 ms or second or third degree AVB, and bradycardia below 60 bpm (SOUZA et al., 2021).

The use of heparin is preferentially indicated for LMWH (enoxaparin), as it does not require laboratory control and determines a lower incidence of thrombocytopenia. The dose is 2 mg/kg/d divided into 2 doses for at least 48 hours in UA/NSTEMI. Enoxaparin should be administered at the time of diagnosis of STEMI in the following doses: for patients under 75 years of age, administer 30 mg intravenously followed by 1

mg/kg SC every 12 hours until hospital discharge; in patients over 75 years of age, do not administer the intravenous dose and start with 0.75 mg/kg SC every 12 hours. Continuous intravenous UFH can be used, especially when an early surgical approach is possible. However, UFH is superior in protecting against reinfarction and post-AMI angina. After angioplasty, there is no longer an indication to maintain full heparinization (DARZÉ et al., 2024).

For patients with coronary heart disease, in an attempt to avoid an increase in atherosclerotic plaques in other vascular territories, it is recommended to maintain LDL cholesterol levels below 100 mg/dL, emphasizing that the recommended goal for patients admitted to hospital units with non-ST-segment ACS is LDL cholesterol levels below 70 mg/dL. Its use is indicated in any condition of proven ACS (PAIVA et al., 2022).

ACE inhibitors should not be started immediately after patient discharge, but it has an impact on the survival of patients at risk of progression to ventricular dilatation. As for those with STEMI and systemic arterial hypertension, diabetes mellitus, maintenance of hypertension, and signs or symptoms of left ventricular dysfunction. It can be started at low doses, from 6 to 24 hours after the event, with rapid progression of the therapeutic dose, according to the patient's tolerance (SOUZA et al., 2021).

Catheterization is a safe procedure, with a mortality rate of 0.1% and morbidity of 5%. It is indicated for candidates for revascularization, patients with recurrence of unstable symptoms, or surgical valvular heart disease. Acute Coronary Syndrome should be performed in cases of troponin positive, dynamic ST changes, heart failure, hemodynamic instability, recent revascularization, ejection fraction <40%, or cardiac arrest survivors. When these criteria are not met, it can be done later for stratification and treatment of coronary disease (FERNANDEZ et al., 2024).

CONCLUSION

Effective management of patients with chest pain in the emergency room is an essential skill that the health team in the in-hospital environment must know how to manage appropriately. To do so, this skill requires ongoing training, robust technical knowledge, and the use of appropriate strategies. The techniques must be adapted to the patient's clinical context, taking into account the results of their electrocardiogram and biomarkers as well as their clinical practice and associated comorbidities, always

prioritizing the patient's safety and stabilization and avoiding as many sequelae as possible (ASUNÇÃO et al., 2020; DO CARMO et al., 2022).

Despite the challenges of patients with acute coronary syndrome, the advancement of technological devices, such as catheterization and angioplasty, and the use of structured protocols have contributed significantly to improving outcomes. The training of health professionals as physicians, and the availability of resources continue to be fundamental pillars for the successful management of patients with acute coronary syndrome (BEATO NETO et al., 2022).

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