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CLINICAL OR INTERVENTIONAL TREATMENT IN ACUTE CORONARY SYNDROME

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Abstract: Introduction: ACS has a wide clinical presentation, ranging from asymptomatic conditions to cardiac arrest, with chest discomfort being the main clinical symptom. Chest pain is a challenge for doctors in the emergency room and accounts for 5 to 10% of visits. Differentiating non-cardiac chest pain from that of cardiac origin requires attention and mastery of diagnostic methods through the patient's clinic and tests such as troponin and electrocardiogram. Given the wide prognostic range, patients must receive a risk classification and, based on these data, receive appropriate therapeutic management. **Goals:** The main objective of this work is to understand the diagnostic methods and procedures applicable to everyday clinical practice, remaining within the recommendations of current scientific literature. **Methods:** This is a narrative bibliographic review. Scientific articles selected from the VHL, PubMed and Scielo databases were consulted. The eligibility of previously selected publications was based on the most recent studies available on the topic of ACS and AMI, preferably from 2019. **Conclusions:** The doctor must have ready access to the main recommendations and conduct of the most relevant guidelines on the subject of ACS/AMI and be able to adapt them to the local reality. This way, this work takes into consideration practical issues, based on current references and with a direct and objective reading on which flow to establish for each patient according to the technical structure of the unit where the patient with acute coronary syndrome was admitted and thus allow decision making between clinical or interventional treatment.

Keywords: "Coronary Artery Disease", "Acute Coronary Syndrome" and "Acute Myocardial Infarction"

INTRODUCTION

Acute coronary syndrome (ACS) has a broad clinical presentation that can range from cardiac arrest, changes in electrical or hemodynamic conduction leading to cardiogenic shock due to myocardial ischemia or even present asymptomatically when the patient arrives at the medical unit. health. However, the main symptom that will lead to the diagnostic and therapeutic cascade of a suspected ACS condition is acute chest discomfort, which can be characterized by pain, pressure, tightness or burning in the precordial region.[1]

The symptom of chest pain is a challenge for the doctor in the emergency room due to an extensive range of possible differential diagnoses between musculoskeletal, gastrointestinal, cardiac, psychiatric and pulmonary disorders, which can present an amount of 5 to 10% of consultations in emergency rooms. emergency care units, with ACS being responsible for 1/5 of these cases of chest pain. Even so, given these significant numbers, 2 to 10% of patients with ACS are discharged without a properly diagnosed diagnosis and may present a poor clinical evolution, without having received adequate diagnosis and therapy, which leads to the need for knowledge of the main causes of chest pain according to emergency doctors.[2]

Acute myocardial infarction (AMI) received a universal definition proposing that myocardial necrosis must occur associated with clinical changes consistent with myocardial ischemia, the diagnosis of which is determined by changes in cardiac biomarkers, preferably ultrasensitive troponin, T or I, given that at least one result must be above the 99th percentile of the reference limit, in addition to symptoms of myocardial ischemia or evidence of myocardial ischemia on the ECG, such as: changes in the ST segment/T wave or new LBBB (left bundle branch block) or wave development Pathological Q, new myocardial kinetic change on imaging study, or coronary thrombus

detected on angiography or a combination of these changes.[1,3,4]

When evaluating a patient with ACS, the emergency department physician will also stratify the risk of cardiovascular ischemic events, based on each patient's clinical and personal variables. To this end, there are several cardiovascular risk scores described in the literature and, among them, the use of the GRACE score (Global Registry of Acute Coronary Events) and the HEART score are recommended by the Brazilian Society of Cardiology (SBC). [5]

Deaths due to AMI have a high prevalence and pathology studies demonstrate that this is the cause of almost half of all out-of-hospital deaths due to cardiac arrest when all ages are considered, with this proportion directly related to advancing age. Another fact is that the relationship between cardiac arrest preceded by chest pain presents an anatomopathological accuracy for diagnosing AMI in almost 100% of case.[6]

Considering the severity of acute coronary syndrome and the need for rapid and precise treatment, as well as recent advances in cardiology in the last five years, there was interest in preparing this bibliographical review in order to make understanding what is recommended more accessible and practical. in current literature.

GOAL

Review the concepts and pathophysiology of Acute Coronary Syndrome and Acute Myocardial Infarction, as well as learn about the diagnostic methods and interventional approaches most applicable to everyday practice, through the recognition of risk stratification methods and decision-making between clinical or interventional treatment, maintaining within the recommendations of current scientific literature.

METHODS

This work is a narrative literature review. We chose this type of review due to the ease of access to the main guidelines, in order to enable a succinct and objective description of the concepts covered in an objective manner.

This way, a search for scientific articles was developed based on the Health Sciences Descriptors (DeCS) with relevant terms such as: "Coronary Arterial Disease", "Acute Coronary Syndrome" and "Acute Myocardial Infarction" and such terms in English "Coronary Artery Disease", "Acute Coronary Syndrome" and "Acute Myocardial Infarction". The databases consulted were: SCIELO, PUBMED and VHL.

The eligibility of previously selected publications was based on the most recent studies available on the topic of ACS and AMI, preferably from 2019, but with an older publication in 2015 referring to an SBC guideline still in force and applicable throughout the national territory and as an exclusion criterion, studies prior to 2015 and/or that did not have their publications in indexed journals were used.

Due to the nature of the study being a bibliographical review, in which there was no risk related to the target audience and professionals involved, the need to submit this work to the ethics committee for research on human beings was waived.

DEVELOPMENT

PATHOPHYSIOLOGY

AMI is most often caused by an absence or decrease in blood flow to a region of the heart leading to myocardial necrosis, generally caused by a clot formed in the epicardial portion of the coronary artery. However, it is known that myocardial ischemia will not necessarily be caused solely by obstruction to blood flow resulting from a clot. Therefore, a

change in the supply-demand ratio of oxygen to the myocardium, in which demand is greater than supply, may result in the same ischemic process. This change can occur in conditions with high heart rate and excessive demand or a significant drop in blood pressure resulting in low supply, which leads to myocardial damage without the presence of obstruction to blood flow resulting from a clot.[3]

Atherosclerosis is the main factor in the development of endothelial dysfunction and, consequently, obstruction of coronary blood flow and myocardial infarction.

Atherogenesis results from a chronic inflammatory and multifactorial disease in which there is an endothelial response to aggression that mainly affects the intimal layer of large and medium-sized arteries. This attack on the vascular endothelium occurs due to several risk factors such as high blood pressure, smoking or dyslipidemia. In the intima of the coronary artery, a lipid core is formed surrounded by protective matrix tissue, forming a stable lipid plaque. Over time and in the presence of lymphocytes with a more inflammatory phenotype, presence and greater release of metalloproteases synthesized by macrophages and migration of smooth muscle cells to the vascular intima that inhibit collagen synthesis, there is a reduction in the matrix tissue leaving the lipid plaque. vulnerable to complications. The dysfunctional endothelium allows the penetration of plasma low-density lipoproteins (LDL) into the intima, where they are retained in the subendothelial space and undergo oxidation. Oxidized, low-density lipoproteins become immunogenic. This is the key process in initiating atherogenesis.[7]

As a result of oxidized LDL, leukocyte adhesion molecules appear on the endothelial surface, attracting lymphocytes and monocytes to the arterial wall. Monocytes reach the lipid nucleus, transform into macrophages and capture oxidized LDL, becoming foam cells, which are the main components of fatty streaks, which macroscopically qualify initial atherosclerosis. This way, activated macrophages begin to release cytokines and proteolytic enzymes, progressing to the atherosclerotic plaque and associated with smooth muscle cells, there will be the formation of the atherosclerotic fibrous cap, which can be stable or unstable. Unstable plaques are associated with the development of ACS and AMI (acute myocardial infarction) and present intense inflammatory activity, have a tenuous fibrotic layer, a lipid and necrotic core with intense proteolytic activity, which predisposes the rupture of the fibrotic layer with exposure of material. highly thrombogenic lipid, leading to thrombus formation and complete or partial obstruction of downstream coronary blood flow.[7]

Atherosclerotic disease can morphologically present its growth in a concentric form, in which the atheromatous plaque will cause stenosis in the coronary lumen, exceeding its expansion capacity and, thus, occluding the coronary lumen, or in an eccentric form, in which extensive plaques can grow in the affected coronary wall without causing symptoms or being noticed on arteriograms. The growth of atherosclerotic plaques in the coronary arteries associated with their compensatory increase, so that the plaques remain accommodated without a decrease in blood flow, is a commonly observed mechanism to prevent luminal stenosis.[8]

DIAGNOSIS

In the emergency service, patients with chest pain must be screened through a brief clinical history, physical examination, performance and medical interpretation of the ECG within 10 minutes of the patient's entry into the service, measurement of biomarkers

(troponin) and application of the HEART risk score. This way, the highest risk patient who requires hospitalization or who needs urgent transfer to the hemodynamics service can be identified earlier.[5]

Biochemical markers are essential in the diagnosis and prognosis of ACS. Therefore, currently, markers are used that are protein constituents of myocardial cells and have no enzymatic function. When these cells are irreversibly damaged, there is a loss of integrity in their membranes and intracellular proteins diffuse into the interstitium, lymphatic vessels and capillaries, which will be identified by specific laboratory tests when they reach minimum concentrations inherent to the diagnostic method used. However, it is still unclear whether the presence of these proteins in the circulation is a factor that indicates a necessarily irreversible injury to myocardial cells.[5]

Therefore, in those patients who present clinical findings compatible with ACS, but in whom the diagnosis of non-ST segment elevation myocardial infarction (STEMI) has not been established, biochemical markers must be used to confirm the diagnosis and establish prognostic possibilities.[4] The proteins that are not present in smooth muscle and are part of the myocardial myofibrillar regulatory complex are troponins. There are three subunits of troponin, troponin T (TnTc), troponin I (TnIc) and troponin C, however the latter is also present in slow-twitch muscle fibers and, therefore, is not considered a specific cardiac biomarker. Therefore, troponin T (TnTc) and troponin I (TnIc) are currently the biochemical markers of choice for myocardial necrosis when AMI is suspected due to their high sensitivity (90%) and almost total specificity (97%), but no biochemical marker is perfectly accurate in determining myocardial damage. There is almost no difference between TnTc and TnIc as they present practically identical clinical information. Therefore, the choice depends on the equipment available in the clinical pathology laboratory.[4] The T (TnTc) and I (TnIc) subunits are specific to the myocardium, with TnIc found only in the myocardium and, alone, has 100% specificity and can be the ideal marker of myocardial necrosis. TnTc units, although highly specific, can be released in other clinical conditions such as rhabdomyolysis, stroke, sepsis, chronic kidney disease, gastrointestinal hemorrhage and hypertensive emergencies, which can lead to false-positive results for ACS, with the association of levels of elevated TnTc in patients without ACS, a poor prognostic factor for those diseases.[9] On the other hand, patients who present with chest pain and undetectable troponin levels, as well as the absence of signs of ischemia on the ECG, presented a minimal risk of AMI or death within a period of 30 days, with a negative predictive value of 99.8%. for myocardial infarction. [10]

Women with ACS have particularities as they present important differences from their clinical presentation and, unlike men, go to the emergency room later after the onset of symptoms, as they often wait to finish housework or do not want to disturb other family members, which leads to a delay of more than an hour in presenting for medical care when compared to men, resulting in a longer time to start treatment.[11]

There are two pathophysiological types of development of myocardial ischemia, classified as type 1 and type 2 myocardial infarction, in which type 1 develops through acute ischemia being represented by ACS and type 2 is caused by oxygen supply-demand prolonged disability due to luminal stenosis caused by atherosclerotic disease with stable plaque.[8]

According to changes in the electrocardiogram, patients with ACS must

be divided into two groups. The first group is made up of patients who present with acute chest pain and persistent elevation of the ST segment for more than 20 minutes. This condition denotes an acute total or subtotal coronary occlusion and the main approach to be taken is immediate coronary reperfusion by primary percutaneous coronary intervention (PCI) or, if not available, fibrinolytic therapy. This makes up the group of acute coronary syndromes with ST-segment elevation (ST-SCST). The other group of patients are those with acute chest discomfort, but without persistent ST segment elevation, in which the electrocardiogram (ECG) may demonstrate persistent or temporary ST segment depression, T wave inversion, flat T wave or even a Normal ECG and make up the group of acute coronary syndromes without ST-segment elevation (NSTE-ACS).).[1,5]

When there is myocardial ischemia associated with the absence of myocardial necrosis, determined by negative biomarkers such as troponin, it is defined that the patient has unstable angina (IA). However, in the initial management of ACS it is difficult to differentiate when we are faced with unstable angina or NSTEACS before the results of myocardial necrosis markers are available and reliable, according to the minimum time expected for myocardial release of these markers and their appearance in systemic circulation. Therefore, both SCASSST and IA must be conducted in a similar way with regard to the therapy used.[5]

It is important that the emergency physician knows that the diagnosis of AMI is clinical and electrocardiographic. Myocardial necrosis markers must not be used for diagnostic purposes in patients with ST segment elevation, as there is evidence that such a procedure is not useful or effective or, in some cases, may even be harmful to the patient (class III, level of evidence C).[4]

RISK STRATIFICATION

According to the SBC, all patients must receive risk stratification by cardiovascular risk scores, which can be classified as high, very high, intermediate or low risk of developing major cardiac events. More than one method must be used and the worst-case scenario is what must be considered for decision-making and medical approach. This is a recommendation with evidence that the procedure is effective (class I), with data obtained through meta-analyses or observational studies (level B).[1,5]

The GRACE cardiovascular risk score presents greater mathematical complexity and, therefore, must be carried out using calculators or specific applications, but it allows for more accurate stratification both at patient admission and discharge, using eight variables such as (1) age in years, (2) heart rate, (3) systolic blood pressure, (4) creatinine levels, (5) Killip classification, (6) cardiac arrest on admission, (7) ST segment shift, and (8) elevation of biomarkers of myocardial injury. This score calculates the risk estimate for the outcomes of in-hospital death at 6 months, 1 year and 3 years and also the risk of death or AMI within 1 year.[5]

The HEART score stratifies the patient for the risk of a major cardiac event (death, heart attack or need for revascularization) within 6 weeks after admission, for patients treated with chest pain. It evaluates variables such as history presented at admission regarding suspicion of ACS, changes in the ECG, age, risk factors and presence of troponin. [5]

When compared to GRACE, the HEART score was better able to distinguish patients at low risk of major cardiac events. Therefore, a patient who presents a HEART score ≤ 3, associated with negative troponin (at an appropriate time), ECG without ischemic changes and no history of coronary artery

disease, can be safely discharged from the emergency department for outpatient reassessment. This conduct has an I/B recommendation [1,5]

The stratifications will define the patient's prognostic expectation but will also help the doctor in making decisions upon the patient's arrival, as patients with hemodynamic instability must have the affected coronary artery opened within two hours, while high-risk patients can receive PCI within 24 hours and intermediate risk within 72 hours. [1,5]

CLINICAL MANAGEMENT

The gold standard treatment for patients with ST-segment elevation myocardial infarction (STEMI) is PCI, in addition to being a therapeutic option for patients with non-ST-segment elevation AMI and stable coronary artery disease.[12] Timely reperfusion of the infarction-related coronary artery has the capacity to save the myocardium in an ischemic process and prevent ventricular remodeling.[13] Therefore, PCI has become the method of choice when aiming for the mechanistic treatment of obstructive coronary disease.[14]

However, although PCI is the gold standard treatment in STEMI, this procedure is not always available in the health network where the patient was admitted or in a timely manner for transporting the patient. Therefore, fibrinolysis and subsequent referral of the patient for coronary angiography is a viable option with unquestionable benefit.[15]

In patients presenting with STEMI with refractory angina or hemodynamic or electrical instability, malignant arrhythmias, mechanical complications, acute heart failure or recurrent changes in the ST segment and its intermittent elevation, an urgent/immediate invasive strategy is indicated.[5,16] Coronary intervention must occur within 2 hours or the patient must be transferred to a service with interventional cardiology available. These are patients classified as very high cardiovascular risk. In high-risk patients, that is, those with positive troponin, dynamic changes in the ST segment and a GRACE score greater than 140, early invasive intervention must be chosen, with a time for coronary approach of less than 24 hours. In cases where the risk is intermediate, exemplified by diabetes mellitus or renal failure, heart failure and left ventricular ejection fraction less than 40%, PCI or previous coronary artery bypass surgery and GRACE score of 109 to 140, invasive intervention can be carried out within a period of up to 72 hours.[1,5]

In patients with STEMI, the approach must be taken in a more dynamic and assertive way, which may be through reperfusion therapy with the use of fibrinolytics, being very important in scenarios where PCI is not available in an adequate time or even in the pre- hospital, always within the time interval established by the literature.

Studies demonstrate that in the absence of absolute contraindication to fibrinolysis and in the presence of criteria that constitute STEMI (symptoms of ACS associated with the presence of persistent elevation of the ST segment in at least two contiguous leads, as well as in cases where there is a new or presumed new LBBB) present class I and level of evidence A, that is, there is evidence that the procedure is safe and effective, with data obtained from multiple randomized studies and/or consistent meta-analyses of randomized clinical studies, thus offering safety to the patient and to the emergency doctor to carry out the procedure, as long as all criteria and indications relating to the method are followed. Therefore, the use of fibrinolytics presents a concrete indication, except in situations of contraindication. [4]

The 5th guideline of the Brazilian Society of Cardiology (SBC), on the treatment of

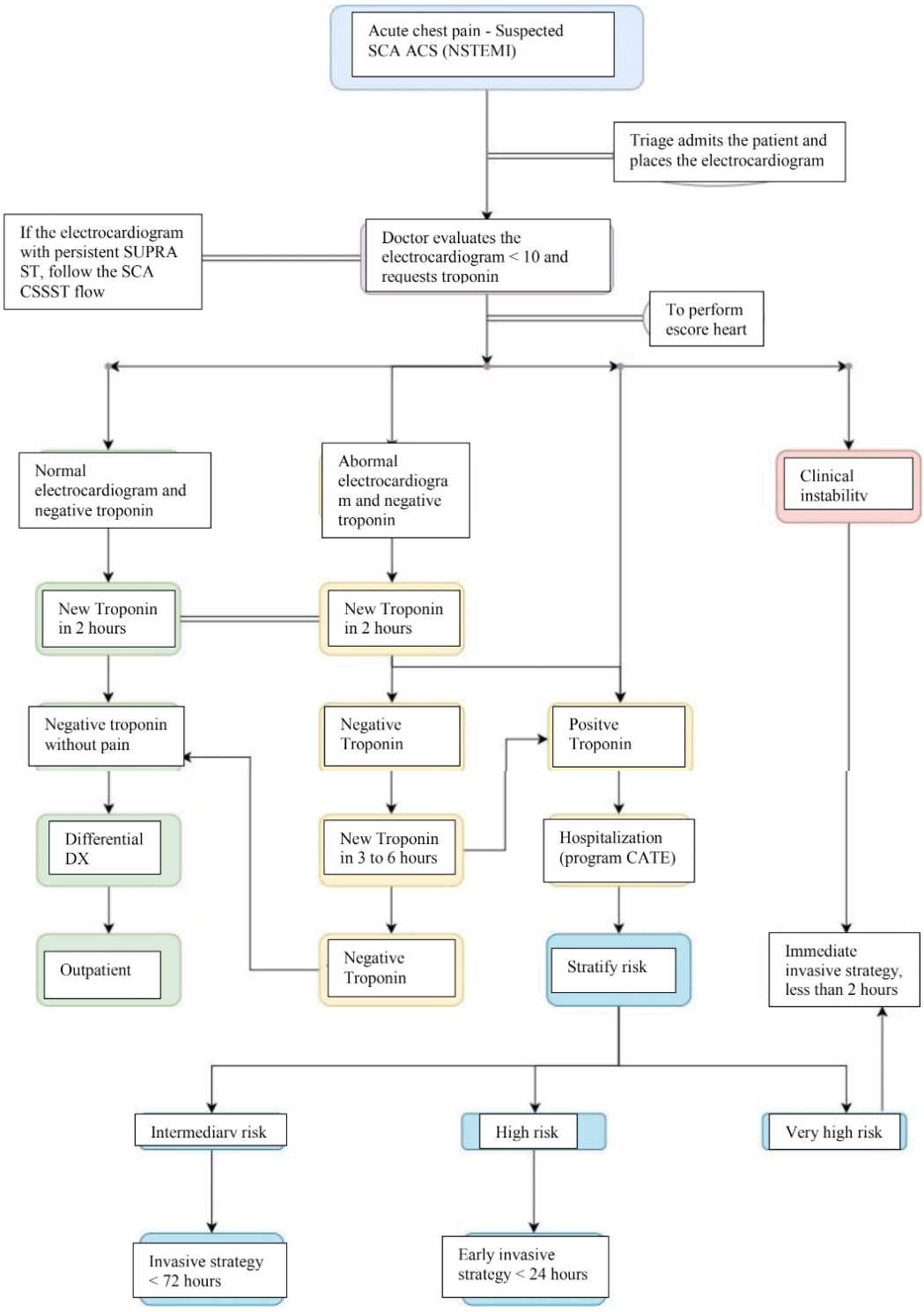

Figure 01: Algorithm for ACS without ST segment elevation.

Source: Adapted from the Brazilian Society of Cardiology and the European Society of Cardiology.

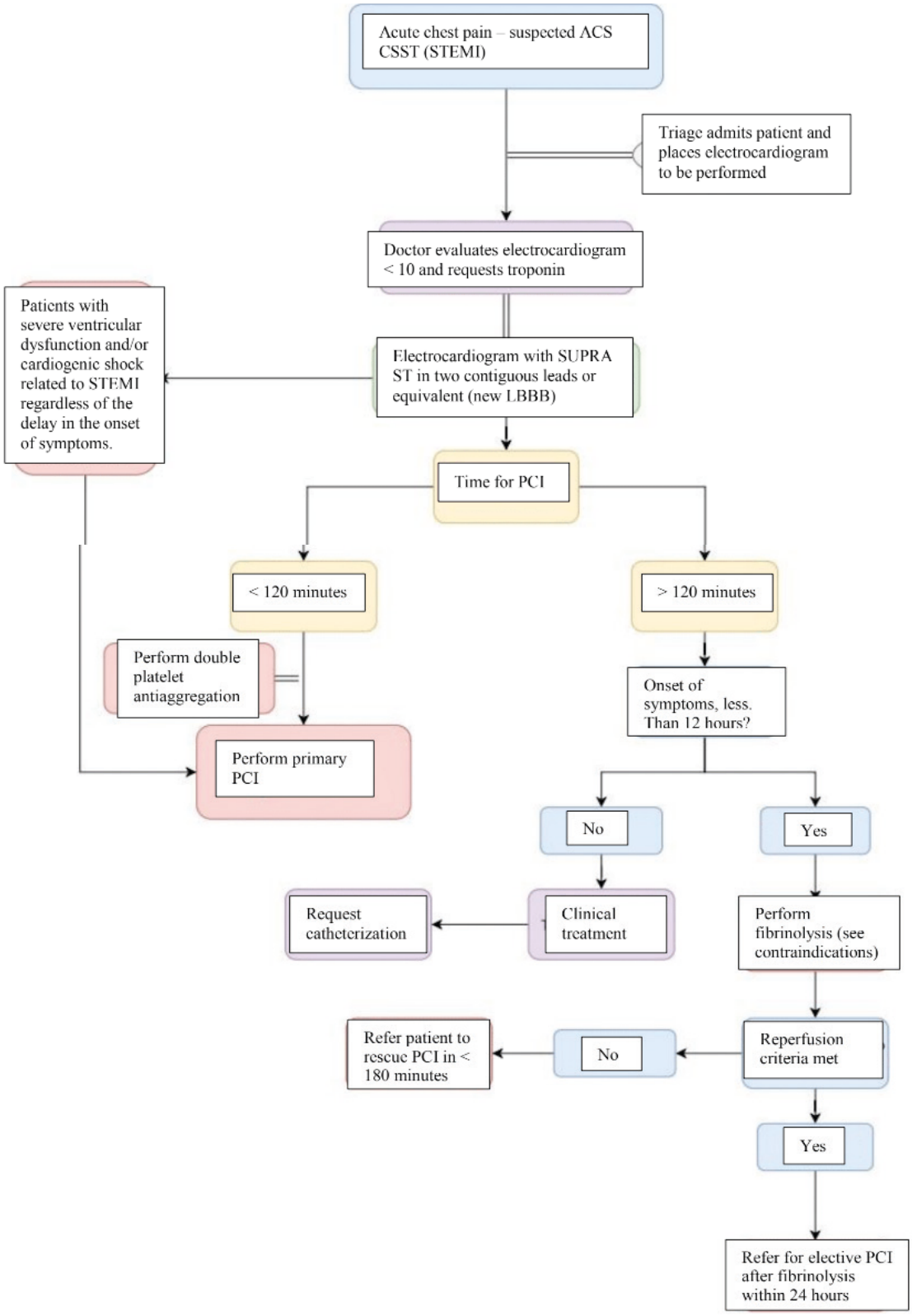

Figure 02: Algorithm for ACS with ST segment elevation.

Source: Adapted from the Brazilian Society of Cardiology and the European Society of Cardiology.

AMI with ST segment elevation, advises the use of fibrin-specific thrombolytics, such as alteplase or tenecteplase, in the first 3 hours after the onset of pain in cases where PCI is not possible, followed by the patient's transfer to an interventional cardiology service within the next 6 to 24 hours. In situations where there is a possibility of PCI, if it is performed in the same unit as the patient's admission, it is recommended that the time for PCI does not exceed 90 minutes, or in case of possibility of transfer to perform PCI in a capable unit, the target time from the patient's arrival at the originating unit to the catheterization is no longer than 120 minutes. In other words, the door-to-needle time (time from admission to the start of thrombolysis) is 30 minutes and the door-to-balloon time (time from admission to PCI) is 90 minutes when in the same unit or up to 120 minutes when there is a need for transfer.[4]

The integration of the two modalities of AMI treatment, through fibrinolysis and primary PCI, called a pharmaco-invasive strategy, is a way of expanding access to coronary reperfusion for infarcted patients in Brazil as it allows an initial approach through the use of fibrinolytics, facilitating logistics for transfer to the tertiary unit qualified for reperfusion therapy in that location.[4]

In the guideline published by the European Society of Cardiology on the management of AMI with ST segment elevation, from 2017, the door-to-needle time is 10 minutes, through the administration of the fibrinolytic tenecteplase as a bolus. The door-to-balloon time is similar to that indicated by SBC.[17]

CONCLUSION

The guidelines, both nationally presented by the Brazilian Society of Cardiology (SBC), and the European one formulated by the European Society of Cardiology (ESC), are very well supported by the literature, with strong and safe recommendations.

However, when put into practice across the vast Brazilian territory and in the face of immense social, geographic and political differences, they demonstrate that our emergency care system is not capable of offering the completeness of published guidelines. Therefore, it is at the discretion of the units, municipalities or administrative regions, according to their technical staff and available resources, to develop specific protocols, taking into consideration, their strengths and weaknesses, but following the recommendations described in the scientific literature.

This way, this work takes into account practical issues, based on current references and with direct and objective reading, presenting in a relevant way the most important details of the medical approach to ACS so that the understanding of the recommendations on clinical or interventional management published in guidelines is clearer, as well as a flowchart suggested for patients presenting to emergency care units with algorithms simplified to the reality of most units (figures 1 and 2). Thus, the doctor can choose the best approach according to the technical conditions of the municipality and/or unit in which he is working without, in any case, deviating from what is recommended by the scientific literature.

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