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CARDIOGENIC SHOCK IN A PATIENT WITH LONG-TERM CORONARY ARTERY DISEASE

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Abstract: Coronary artery disease, in developed countries, is a major cause of sudden death among men and women, accounting for about one third of all deaths. It occurs in 2% to 9% of people aged 20 years or older. The mortality rate tends to increase proportionally with age, being higher for men than for women. After the age of 55, the mortality rate among men decreases, and that of women increases progressively. In this literature review, we associate the clinical picture of a patient in cardiogenic shock due to coronary artery disease and its complications. We explore the management of symptom progression in the cardiologic emergency. The central development of the article was built based on the case report published in the book: Case Files Cardiology, by the publisher: McGraw-Hill Medical Publishing, case 4, page 60, edition 1. In the case in question, a patient was identified, 55-year-old male with a long history of coronary artery disease, admitted to the ICU with hypotension, followed by 24 hours with intermittent chest pain. An intravenous drug solution of nitroglycerin was administered to treat heart failure, leaving him free of chest pain. On the second day of hospitalization, the patient developed angina pectoris, dyspnea, and altered mental status. Data collection showed acute decompensation in his general condition. Cardiogenic shock was observed, due to hypotension, hypoperfusion, lowered mental status, and low oxygenation of the extremities. On physical examination, B3 heart sound was evident. Based on the clinical picture of this patient, we can observe the worsening of morbidity, through the description of the constant hemodynamic evaluation to detect the observed heart failure. We demonstrate the undeniable thorough and accurate assessment of patients with heart disease, in physical and laboratory examinations, in patients in cardiological emergencies.

Keywords: Cardiac emergency; Cardiac insufficiency; Coronary artery disease.

INTRODUCTION

Shock is a state caused by circulatory deficiency, therefore, due to blood shortage, there is a decrease in the supply of oxygen to cells and tissues, which generates hypoxia and necrosis in the affected areas. That said, it is valid to show that cardiogenic shock is the consequence of cardiac pump failure that results in reduced cardiac output. (GAIESKI; MIKKELSEN, 2022).

Furthermore, it can be noted that cardiogenic shock is one of the main reasons that lead to death in patients who have a history of acute myocardial infarction (AMI), in addition to the fact that 5 to 10% of AMI cases are complicated by cardiogenic shock (CS). When it comes to the statistics of the etiology of shock, acute myocardial infarction is hegemonic with 81% of cases (VAHDATPOUR et al., 2019), due to left ventricular failure, being more common in men (without significant discrepancy) and patients with 60 years of age or older (MALAKAR et al., 2019; MENDES et al., 2022), in addition to causing more than 2.4 million deaths in the United States alone and affecting more than 7 million people annually (REED et al., 2017). It is also important to highlight that CS mainly affects the female population, with a higher prevalence in patients from Asia/Pacific Islands and people over 75 years of age (VAHDATPOUR et al., 2019), which may worsen of the condition in the presence of hypovolemia, sepsis, severe valvular heart disease, bradyarrhythmias and heart blocks, tachyarrhythmias (atrial fibrillation, supraventricular or ventricular tachycardias. (FALCÃO, 2017).

Furthermore, it is known that it can be categorized into three types, the first of which is arrhythmic, normally caused by tachyarrhythmias and bradyarrhythmias; the second is mechanical, usually caused by abscesses, valve insufficiency and aortic dissection; while the third is cardiomyopathy whose main cause is coronary artery disease, that is, when there is occlusion of the coronary arteries (CAD). Emphasizing this disease, it is essential to mention that the prevalence has varied greatly according to the location, geography, ethnicity and gender of patients (GO et al., 2014). Still, studies in the United States showed a higher incidence in: patients over 65 years old, people residing in urban centers and males (MALAKAR et al., 2019). Another important point is the risk factors. Among them, smoking, high cholesterol levels, directly linked to endothelial dysfunction, promoting the reduction of nitric oxide and increasing the formation of free radicals together with endothelial activity, culminating in the impairment of the vasodilator capacity, thus, generating arterial hypertension, another important risk factor for CAD. A positive factor is that with a change in lifestyle, the patient with high cholesterol level usually shows significant improvement and, consequently, a lower risk for coronary artery disease (MALAKAR et al., 2019).

METHODOLOGY

The preparation of this literature review article had as the general and specific objectives, the determination of starting points for the research. Such as: "What are the results of the complication of coronary artery disease and its evolution to cardiogenic shock? "; "What are the results of these complications at the systemic level? "; "What is the course/prognosis of the disease when treated? What is the course/prognosis of the disease when untreated? "; "To describe the pathophysiology of coronary artery disease — the entire process, from causes, such as arterial obstruction due to atherosclerosis/

uncontrolled SAH. "; "How do these changes lead to cardiogenic shock?"; "Explain the pathophysiological mechanism of cardiogenic shock — How does it start?"; "How is the blood flow of the patient in cardiogenic shock presented?"; "How to manage the evolution of cardiogenic shock in the emergency?"; "What happens to the arteries in terms of expansion/compliance?"; "Which arteries can suffer cardiogenic shock from coronary artery disease? Are there arteries that are usually more affected? If yes, why?". Other guiding methods for the research were the bibliographies used as a basis for the construction of the article, as well as recent publication dates and updated sources up to the moment of this publication.

RESULT AND DISCUSSION

Regarding the pathophysiology of cardiogenic shock, there is a decrease in Cardiac Output (CO), which in most cases is due to poor myocardial contractility. However, it can also be due to diastolic dysfunction especially in patients with heart conditions such as myocardial ischemia and hypertrophic cardiomyopathy. Heart function can be affected by the course of cardiogenic shock, and the means of compensatory mechanisms of this shock have a deleterious effect from the onset of the dysfunction. (MEJÍA, 2020.)

Cardiogenic shock usually leads to left ventricular dysfunction, generating an increase in systemic vascular resistance (RSV) and consequent increase in afterload, in order to improve circulation. However, this increase in left ventricular effort at the end of systole causes several responses that further damage the condition, such as rigid walls and decreased ventricular elastance. In addition, an inflammatory process occurs, recruiting pro-inflammatory substances such as TNF, IL-6 and catecholamines, causing irreparable damage to cardiomyocytes and causing a greater increase in SVR, decreasing cardiac output and consequently decreasing tissue perfusion. Acute myocardial infarction (AMI) is among the main causes related to coronary artery disease that can lead to cardiogenic shock. Other contributors, in addition to AMI, are: Congestive heart failure, heart valve injuries, tachyarrhythmias and bradyarrhythmias (both atrial and ventricular), stunned myocardium after cardiac arrest, viral and alcoholic heart diseases. (CAMACHO, 2019).

The decrease in cardiac output also leads to the release of catecholamines, which results in vasoconstriction of peripheral arterioles and increased levels of vasopressin and angiotensin II, as a result of which the load increases, which worsens myocardial function. As a result of this process, there is activation of the neurohumoral cascade, with increased retention of sodium, water and volume overload, culminating in pulmonary edema. This cycle is hemodynamically characterized by a decrease in CO, an increase in preload and peripheral vascular resistance, differing from other types of shock because it does not have a compensated initial phase. (MEJÍA, 2020.) In preload, there is the Frank-Starling law that establishes the length of myocardial fibers immediately before contraction, relating it to the volume of blood that reaches the Right Ventricle (RV) at the end of diastole. Some factors influence this preload, such as blood volume, blood volume distribution, ventricular filling and ventricular function. In addition, RV preload is measured through a catheter located in the right atrium or superior vena cava, and left ventricular preload is assessed by measuring pulmonary capillary pressure (PCP) with a Swan-ganz catheter. In contractility, the strength of the myocardial fibers, when contracted, can be evaluated through cardiac output. (MEJÍA, 2020.)

Finally, there is the afterload, which is the difficulty of the left ventricle to eject blood.

This translates into the addition of causes that oppose ventricular contraction. For the left ventricle, ventricular wall thickness, aortic pressure, and aortic impedance are some of these factors. Depending on the etiology, the 3 factors mentioned above may favor the onset of Heart Failure (HF). Low cardiac output causes tissue hypoperfusion in various organs with hypotension, which consequently leads to hypoxemia, anaerobic metabolism and lactic acidosis. If not treated correctly, it can generate a refractory cycle to death. (MEJÍA, 2020).

The endothelium gives functional features to the coronary arteries, together with vascular smooth muscle cells and parts of the connective tissue in the arterial walls. It works by regulating vagal tone, modulating inflammation, promoting and inhibiting neovascular growth, and modulating platelet aggregation and coagulation. This way, the endothelium performs the synthesis of vasoconstriction and vasodilation factors. Nitric oxide (NO) is one of the factors responsible for vasodilation, or vascular relaxation, of greater importance arising from the endothelium. (PINHO, 2010).

In healthy people, vasodilator tone is moderate and constant, maintaining vascular homeostasis due to NO. If basal NO production ends/decreases considerably, vasoconstriction occurs. The reduction in NO results in decreased blood tissue perfusion and is a predisposing factor for thrombus formation. High NO production, on the other hand, causes vasodilation and can lead to shock. The change in the production or reduction of NO can commonly result from diseases such as Dyslipidemia, SAH (systemic arterial hypertension) and atherosclerosis. Thus, endothelium dysfunction, which may be a consequence of NO reduction, helps in the emergence of CAD (coronary artery disease). The presence of the disturbance of the endothelium is demonstrated in the Angiography before the clinical picture appears, characterizing the Atherosclerosis. (PINHO, 2010).

Cardiogenic shock, through interruption or considerable decrease in blood flow, mainly affects the arteries that supply blood to the myocardium and the heart as a whole. The shock will depend on the damage to the heart muscle, resulting in a decrease in the pumping capacity of the heart muscle, leading to the extension of the damage to the coronary arteries in question, which, in turn, are the most affected vessels in coronary artery disease. The disturbance of blood flow provides a positive feedback on the systole/diastole change. Some medications to control CAD's can be managed, such as beta-blockers and angiotensin converting enzyme (ACE) inhibitors. These drugs work by reducing the strain on the heart muscle, helping the muscles to maintain a regular rhythm. The heart, when injured, may increase in size to compensate for decreased systemic pumping. However, it can result in cardiac rhythm abnormalities, such as bradykinesia, ventricular tachycardia and ventricular fibrillation, which are at risk for cardiogenic shock. (FONSECA, 2016).

The shock goes through phases, in the initial phase the patient's condition is reversible and compensated, the absence of oxygen supply is compensated through increased heart rate and increased contractility of the heart. In the compensated phase, there are the classic signs and symptoms of shock, but hypotension has not yet been installed. If the condition is not reversed, it progresses to decompensated shock with the classic signs and symptoms of shock, hypotension and altered state of consciousness. If the patient's condition is not modified, it may progress to irreversible damage to several organs due to tissue hypoxia, resulting in multiple organ failure and death. (AULER JR, 2020).

After the first evaluation of the patient and confirmation of the classic signs and symptoms of cardiogenic shock (CC), it is necessary to start hemodynamic monitoring (MH). In MH, the first step is to determine the cardiac output (CO), where a reduction is expected (↓CO) and then assess the blood volume. In addition to this, it is essential to investigate the decompensating factor, the cause of cardiogenic shock. (VAN DIEPEN, 2017). In clinical practice, it is suggested that all patients with CHD be evaluated with complementary imaging tests (ECG, chest X-ray and echocardiogram) with the specific purpose of understanding the dominant mechanism responsible for acute hemodynamic instability. If an acute aortic syndrome or pulmonary embolism is suspected, a CT scan or transesophageal echocardiogram is appropriate. Echocardiography is the gold standard in the diagnosis and follow-up of patients with CHD. It expresses important information about ventricular function, estimates preload values, investigates myocardial structures and excludes mechanical causes in acute coronary syndrome/acute myocardial infarction (ACS/AMI). Suggested laboratory tests include a complete blood count, electrolytes, creatinine, liver function tests are necessary as they are frequently affected by poor perfusion, arterial blood gases and lactate are part of the hemodynamic parameters of evaluation, and serum levels of cardiac troponin. (VAN DIEPEN, 2017).

The prognosis of the treatment of coronary artery disease depends on some associated factors. Studies carried out in Brazil have shown that the highest rate of morbidity and mortality in the country occurs with people from lower social classes. Studies carried out in developed countries have shown higher morbidity and mortality rates in patients with a greater number of obstructed arteries compared to patients without obstruction (>50%). Lack of access to specialized treatment after CAD detection, including more advanced surgical approaches, increases the prognosis discrepancy when comparing higher and lower social classes. Access to tertiary care, both in developed and underdeveloped or developing countries, proves to be a better prognostic factor due to the technological advances that are accessed in these areas. The treatment of CAD in primary and/or secondary care only influences a worse prognosis, according to the study. (BRUNO, 2021).

CONCLUSION

In view of what has been discussed, it is clear that Coronary Artery Disease (CAD) is considered in some countries as a non-communicable epidemic. This is due to the fact that it is associated with certain external factors, such as: lifestyle habits, sedentary lifestyle, smoking, alcohol consumption, overweight, Systemic Arterial Hypertension, diabetes and poor diet. Thus, when these factors are potentiated, and when the individual already has certain genes that favor this issue, the risks are quite high for the development of the disease and complications.

In the meantime, it is clear that before the patient is affected by cardiogenic shock, the body emits some signals. Thus, Primary Primary Care, as the gateway for individuals to Public Health, has the function of accompanying users, requesting tests, assessing cardiac risks and reducing morbidity and mortality. When cases are more complicated and need the evaluation of a cardiologist or cardiology surgeon, for example, the PHC physician has the role of referring the patient to the secondary level of health care.

During the development of this study, it was observed that urgency and emergency services do not always have the necessary

equipment to perform tests such as electrocardiogram, echocardiogram or specific tests that serve as a more accurate assessment of the case. Many of the patients who are victims of Cardiogenic Shock spend days waiting for transfer to have access to a specialist doctor and specific exams such as tomography and resonance. This way, all this waiting only contributes to the progression of the case and increased risks.

In addition to the risk factors mentioned above, the senescence process itself is something that poses greater risks to the patient. This is because, during the natural aging process, there is a decrease in elasticity and contracture of the artery wall, an increase in cardiac output, in addition to, in partnership with pathological conditions (senility process), being responsible for increasing the risks of DAC development. Therefore, during the anamnesis and physical examination, the physician must carefully observe the personal pathological and physiological history that are collected, in addition to requesting tests in order to verify or rule out a diagnostic hypothesis.

This way, CAD is indeed a Public Health issue and needs mechanisms to reduce the risks and vulnerabilities of patients. In cases of patients who are already at greater risk or who have already developed CAD, lifestyle change (SEM) is essential in this process, both to mitigate the risks of a recurrence and to remove the patient from the risk group.

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