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COMPARATIVE ANALYSIS OF SYNCOPE

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Abstract: Syncopes are common medical conditions characterized by sudden and transient loss of consciousness, together with diffuse cerebral hypoperfusion, associated with reduced blood flow to the brain and loss of postural tone with spontaneous recovery. There is a wide variety of studies that seek to understand and classify syncope, due to the wide scope of its origins, consequences and manifestations. However, the best-known classification divides them into three main types: Cardiogenic, Orthostatic and Reflex. Each of them contains subtypes, which can differ by etiology, most affected population, symptoms and treatment. Examples of these are Hypotensive, Neurogenic and Vasovagal syncope. These classifications help in the diagnosis and development of appropriate therapeutic approaches for each patient.

Studies indicate that syncope, especially Vasovagal, has a correlation with family history, and may be hereditary and possibly explained by some yet unknown genetic component.

Cardiogenic syncope is fainting caused by problems in the cardiovascular system. Elderly people, people with chronic illnesses, people taking medications that affect blood pressure or heart rate, and patients with heart disease are most affected by this type. Its main subdivision concerns arrhythmogenic syncope, which can affect people of any age group. This is caused by a cardiac arrhythmia, that is, a change in the heart's rhythm and depends on the type of arrhythmia present in the patient - bradycardia (slow or absent beat), tachycardia (fast and irregular beat) or heart block (interruption in electrical conduction). from heart). Bradycardia arrhythmia can cause an episode of syncope due to a reduction in heart rate (< 30 or 35 bpm) and, consequently, a reduction in cardiac output.

Although opposite, tachycardia (> 150 or 180 bpm) also results in a reduction in cardiac output, since there is a decrease in ventricular filling time and consequently a smaller volume of blood being pumped around the body. Finally, heart block, which involves high-grade atrioventricular blocks or structural blockages caused by cardiovascular diseases. In this group we have valve diseases, hypertrophic cardiomyopathy, atrial myxoma, pulmonary embolism and pulmonary hypertension, cardiac tamponade, coronary syndrome and finally acute aortic dissection. Among these causes, the two with the greatest population reach are aortic stenosis and hypertrophic cardiomyopathy (HCM). The first often leads to compensatory peripheral vasodilation induced by physical exercise, since the obstruction prevents the increase in cardiac output, and thus results in a drop in systemic blood pressure and cerebral hypoperfusion. HCM causes syncopal episodes in approximately 25% of patients with this disease, due to abnormality and atrioventricular conduction blocks, myocardial ischemia during exertion and subaortic obstruction.

In addition to arrhythmogenic syncope, there are two other subdivisions that are part of cardiogenic syncope, they are: neurogenic syncope and hypotensive syncope. Neurogenic occurs when there is a failure in the nervous system responsible for controlling blood pressure and heart rate, which can lead to a previously seen scenario of arrhythmia. This can be caused by autonomic disorders, such as dysautonomia, or by neurological problems, such as epilepsy. On the other hand, hypotensive syncope is caused by an abrupt drop in blood pressure, usually related to dehydration, medication use, or heart failure. Complications of syncope include injuries from falls, brain injuries from lack of oxygen, psychological trauma and, in rare cases, sudden death.

Although there are differences between the

types of syncope, the symptoms presented in the cases described may include: dizziness or vertigo, blurred vision, weakness, sweating, nausea, irregular heartbeats and fainting. Treatment of cardiogenic syncope depends on the underlying cause, and may include the use of a pacemaker (a device implanted in the chest that helps regulate the heart rhythm in patients with bradycardia), a defibrillator (a device implanted in the chest that helps stop serious arrhythmias), lifestyle changes, such as avoiding the use of certain medications or changing your diet to help control blood pressure, and treatment of underlying conditions that may worsen the trigger for syncope.

Orthostatic syncope is a type of fainting that occurs due to a sudden drop in blood pressure in a situation where a person quickly changes from a sitting or lying position (decubitus) to an upright position (orthostatic). This sudden change can trigger a response from the body that results in a temporary decrease in blood flow to the brain, leading to loss of consciousness. There are two main classifications for orthostatic syncope: neurogenic and non-neurogenic. The first occurs due to a dysfunction in the autonomic nervous system, which is responsible for regulating blood pressure and blood flow. This dysfunction can result in an inadequate response by the body to postural changes, leading to a sudden drop in blood pressure and inadequate cerebral perfusion. Non-neurogenic orthostatic syncope occurs due to causes unrelated to the autonomic nervous system. The most common generators include excessive blood loss and dehydration (hypovolemia) or the use of certain medications that can cause orthostatic hypotension. Among the drugs that can contribute to syncope are diuretics, antidepressants and antihypertensives.

Orthostatic syncope has a higher incidence in certain population groups. Elderly people, people with chronic illnesses, patients taking medications that affect blood pressure, and patients recovering from serious surgery are more likely to experience sudden drops in blood pressure when changing positions. The symptoms most frequently experienced by patients with syncope include dizziness or vertigo when standing up quickly, blurred vision, weakness, paleness, sweating, nausea and fainting. These symptoms reflect the temporary decrease in cerebral blood flow and the lack of adequate oxygenation to the brain during postural change. Treatment of this syncope aims to treat the underlying cause, some options include changing lifestyle, increasing fluid intake to avoid dehydration and avoiding sudden changes in position. Physiotherapy plays an important role in treatment as it can help improve postural control and increase orthostatic tolerance. Additionally, the use of compression stockings may be recommended to improve venous return and maintain stable blood pressure. There are also medications that treat orthostatic hypotension, controlling fainting episodes and improving symptoms, such as Fludrocortisin, Midodrine and Drocidopa. Reflex syncope, also known as neurally mediated, is fainting that occurs as a result of a reflex response generated from the autonomic nervous system. These syncopes can be classified into three main types: Vasovagal, Situational and Careotid Syncopes.

Vasovagal syncope is the most commonly seen form in clinical cases, affecting approximately 35%-39% of the population. It occurs mainly in adolescent women, between 12 and 30 years of age. This condition is triggered when the sympathetic nervous system, associated with communicating chemoreceptors of the vagus nerve, is inappropriately activated in response to emotional stimuli, such as fear and anxiety, or physical stimuli, such as pain

or temperature extremes. This results in a sudden drop in blood pressure and blood flow to the brain. The pathophysiology of Vasovagal Syncope, although not completely understood, is characterized as sympathetic inhibition with consequent bradycardia and relative loss of peripheral vasoconstriction, together with vagal stimulation, resulting in hypotension. The reduction in blood pressure levels induces a state of total cerebral hypoperfusion, damaging the level of consciousness. The diagnosis of patients with Vasovagal syncope (VVS) begins with anamnesis and physical examination and can be confirmed through the "tilt-test" or "orthostatic tilt test (IT)". This test is based on observing the patient's blood pressure and heart rate over an extended period of time. The test is carried out in two phases: passive and active. In the first phase, the patient is placed on a stretcher, in which he remains restrained by the feet, waist and chest, in the supine position for around 10 to 20 minutes and, subsequently, is abruptly tilted to a position with an inclination of 70 degrees and remains like this for another 40 minutes. If the patient does not trigger vagal reflexes during the first phase, the second stage is advanced, in which 1.25 mg of Isordil is administered sublingually to increase the patient's awareness of vagal reflexes. It is important that the patient fasts for 4 hours before the exam and, subsequently, the patient is not allowed to drive. It is also recommended that the patient consume water and food before leaving the establishment, in order to regain normality of their vital signs.

Unlike Vasovagal syncope, situational syncope occurs in response to a specific trigger, usually associated with increased internal pressures in the body. These triggers may include activities such as urination, swallowing, defecation, coughing, post-exercise, and post-laughter. During these actions, there is a temporary increase in pressure in the chest or abdominal region, which can lead to a decrease in blood flow to the brain, resulting in fainting. This form of syncope can affect people of all ages, although it is more common in adults. Finally, there is carotid syncope, which occurs when there is a decrease in blood flow to the brain due to compression of the carotid arteries, located in the neck.

Compression of these arteries can be caused by factors involving inadequate posture, tightness in the region, wearing tight clothing around the neck or even external pressure on the carotid arteries. Carotid syncope is the least common among reflex syncopes. Symptoms of reflex syncope may include dizziness, darkened vision, feelings of weakness, sweating, nausea, irregular or fast heartbeats and fainting itself. Treatment for reflex syncope may include lifestyle changes such as avoiding known triggers, increasing fluid and salt intake, and avoiding sudden changes in position. In more severe and refractory cases, when other measures are not effective, pacemaker implantation may be considered to help regulate the heart rhythm. Additionally, medications may be prescribed to treat vasovagal syncope, such as beta-blockers, serotonin reuptake inhibitors or fludrocortisone, and the use of compression techniques, which may be used to treat carotid syncope, such as carotid sinus compression or electrical stimulation of the carotid sinus, to restore cerebral blood flow.

In short, syncope is the result of varied spectrums, resulting in similar symptoms and signs such as blurred vision, dizziness and fainting. They occur in different types of populations, as seen throughout the analysis. Each variation must have a specific diagnosis and treatment, in order to be efficient in the case. It is therefore important to recognize that each patient will express their symptoms individually, requiring treatment adapted to their needs and complaints. Furthermore,

episodes can be sudden and often random, resulting in difficult early diagnosis. This fact can make ideal treatment unlikely, having to act quickly and often unpleasantly for the patient. Therefore, we are looking for ways to discover syncope before this acute moment of symptoms, a possibly achievable way is to search for neurotransmitter and syncope signaling substances previously, since it has been proven that there is a genetic-hereditary relationship between them.

Keywords: Syncope; classifications; target population; symptoms; treatment.

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