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TOTAL ATRIOVENTRICULAR BLOCK IN SENESCENT PATIENT – CASE REPORT

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Abstract: Atrioventricular blocks (AVB) or atrioventricular (AV) conduction disorders occur when atrial impulses are delayed or fail to reach the ventricles. Total AVB (TAVB) is characterized by the absence of correlation between atrial and ventricular electrical activity, which is translated on the electrocardiogram (ECG) by P waves unrelated to the QRS. It is among the most frequent cardiac arrhythmias, affecting mainly the elderly population. Its treatment depends on the etiology, being performed, in most cases, the definitive pacemaker implantation. Among the various etiologies for the occurrence of AV conduction disorder, we can mention: inflammatory, metabolic/endocrine, drug, hereditary/congenital, infiltrative, neoplastic/traumatic causes, coronary artery disease (CAD), acute myocardial infarction, autoimmune and idiopathic progressive fibrosis, as the most common representative among the degenerative causes. The present work brings a case report of a female patient, 71 years old, hypertensive, type II diabetic, insulin dependent, with chronic CAD and chronic kidney disease and former smoker. On the electrocardiogram (ECG) at rest, on admission he showed AVB. The etiology, clinical manifestations, the importance of diagnosis based on complementary methods, treatment and recommendations are explored, showing resolubility as the main factor in the quality of life of people affected by this disease.
Keywords: Atrioventricular block, Case report, Cardiology.

INTRODUCTION

Ventricular atrial blocks (AVB) or AV conduction disorders occur when atrial impulses are delayed or fail to reach the ventricles. The normal electrophysiological feature of the AV nodule is termed "decremental conduction." This property refers to the reduction of the conduction velocity of

the electrical stimulus in the AV nodule and can be evaluated by means of the PR interval on the conventional electrocardiogram (ECG). This interval is considered normal in adults when they are between 120 and 200 ms and depends greatly on the age and heart rate (HR) of the patient. Anatomically, these delays can be located in the AV node itself (nodal block), in the His-Purkinje system (SHP - intra-His block) or below it (infra-His block). Nodal delays usually present with narrow QRS complexes (< 120 ms) and have a good prognosis (MARTINELLI *et al.*, 2007; LOSCALZO, 2013).

Nodal blocks are classified as: first degree, second degree, high degree and third degree. In first-degree AV block, there is a delay in the AV node, manifested by a prolonged PR interval. Second-degree AV block, Mobitz type I, is defined by progressive prolongation of the PR interval with blockade after a P wave. Mobitz type II is characterized by abrupt conduction failure after a P-wave without prolongation of the preceding PR. In high-grade or advanced AV block, which is a form of second-degree AV block, multiple or successive unconducted P- waves are observed. (CECIL; GOLDMAN, 2011). Finally, we have the third-degree block or total atrioventricular block (TAVB), where the stimuli of atrial origin cannot reach the ventricles and depolarize them, causing a focus below the blockade region to assume the ventricular rhythm. Thus, there is no correlation between atrial and ventricular electrical activity, which translates into ECG by P waves not related to QRS (REY, 2007; BRIGNOLE *et al.,* 2013).

Among the various etiologies for the occurrence of AV conduction disorder, we can mention inflammatory, metabolic/ endocrine,pharmacological, hereditary/ congenital, infiltrative, neoplastic/traumatic causes, coronary artery disease, acute myocardial infarction and idiopathic progressive fibrosis, which is the most common representative among degenerative causes. This aging process begins in the fourth decade of life, and can be accelerated by atherosclerosis, systemic arterial hypertension (SAH) and diabetes mellitus (DM) (HARRISON *et al.*, 2013).

The symptoms of patients with AVB are variable, often asymptomatic. Symptoms of tiredness, asthenia, palpitations, and low cerebral blood flow, such as dizziness, presyncope, and syncope, may be seen depending on the patient's heart rate, ventricular function, position, and activity at that time.

To be the one of intranodal AV localization with benign evolution, requiring outpatient follow-up, without specific therapeutic need. However, when the blocks are of more advanced degrees and are located in the SHP (intra – His or infra - His), they are accompanied by symptoms of cerebral claudication inducing more frequently symptoms of acute cerebral ischemia requiring characterization by ECG or electrophysiological study, because treatment with the implantation of a definitive pacemaker (MP) is mandatory (CONSOLIM-COLOMBO *et al.*, 2015).

The diagnosis of AVBs should be performed with the aid of the 12-lead ECG and preferably performed with long leads, 24-hour Holter and Electrophysiological Study (EEF), the latter being little used because it is an invasive test and not available in all locations, despite being the most complete method to assess the level and severity of the blockade (CONSOLIM- COLOMBO *et al.*, 2015).

Regardless of the type of arrhythmia, the goal of treatment is to ensure adequate cardiac output, individualizing the approach according to the symptomatology, frequency and risk of evolution to more severe arrhythmias (SOUZA *et al.*, 2008).

The pharmacological therapeutic arsenal is poor and ineffective, being used more in emergency units, while assessing the need for provisional PM (transcutaneous or transvenous). The drugs that can be used are atropine, isoproterenol, dopamine and theophyl line. The 1st degree AVB usually do not require specific treatment due to the good evolution. Type I 2nd degree AVB also do not require treatment, unless very symptomatic or if the blockade occurs in the PHS, in which there is a need for definitive MP. In the BAV of 2nd degree type II, in the BAV of 2nd degree 2:1 and in the BAV of 3rd degree, the definitive MP is mandatory (CONSOLIM-COLOMBO *et al.*, 2015).

This study aims to report a clinical case of a 71-year-old patient hospitalized in the coronary care unit of a medium-sized hospital, located in the city of Juazeiro - Bahia, with a diagnosis of BAVT, identify the etiology, discuss its clinical manifestations, importance of diagnosis based on complementary methods, treatment and recommendations, evidencing resolvability as the main factor in the quality of life of people affected by this disease.

CASE REPORT

Female patient, I.G.S., 71 years old, from the city of Canudos with a history of chest pain in tightness radiating to the left upper limb, associated with cough, runny nose and fatigue. She was admitted to the Intensive Care Unit in a medium-sized hospital in the city of Juazeiro – Bahia. She was hemodynamically stable, without vasoactive drugs, normotensive and bradycardic, breathing in room air, with good saturation, comfortable breathing pattern.

Patient known to be hypertensive, with type II diabetes mellitus, insulin-dependent, non-obstructive chronic coronary artery disease, chronic kidney disease and former smoker. On physical examination, she was in good general condition, conscious, oriented, pale, afebrile and hydrated. Cardiac auscultation was unchanged, as was pulmonary auscultation. Vital signs: heart rate: 48 beats per minute; respiratory rate: 18 inspirations per minute and blood pressure: 148 x 76 mmHg. The following medications were used at home: levanlodipine 5 mg/day, hydralazine 50 mg 8/8 hours, spironolactone 50 mg/day, furosemide 40 mg/day and simvastatin 40 mg/day. Laboratory tests, electrocardiogram (ECG), transthoracic echocardiogram (TTE) at the bedside, echocardiogram under pharmacological stress and cardiac catheterization (CATE) were requested.

The results of laboratory tests were: negative troponin, urea: 185 mg/dL, creatinine: 2.9 mg/dL, glycated hemoglobin: 7.9 g/dL, blood gas analysis: pH 7.41; PCO2 33.2; PO2 65.5; HCO3 21.9; SPO2 93%.

The 12-lead ECG showed: normal axis, total atrioventricular block, heart rate of 36 beats per minute, ST-segment alteration, T-wave inversion (figure 1).

In the TTE performed at the bedside, it was evidenced left ventricle with preserved global and segmental systolic function, moderate mitral valve insufficiency, inferior vena cava with inspiratory collapse of 50% (normal).

The stress echocardiogram was positive for myocardial ischemia. Cardiac catheterization showed a 50% lesion in the proximal anterior descending coronary artery and 40% in its middle third, marginal artery with a 30% proximal lesion and the right coronary artery with a 20% lesion in its middle third.

The therapeutic approach adopted was the implantation of a definitive pacemaker.

DISCUSSION

The analysis of the case report brings information that corroborates with the literature. Previous studies demonstrate that advanced age can be a predictor for disease in the cardiac conduction system and, consequently, with the possibility of future need for artificial electronic stimulation (CARRIÓN-CAMACHO *et al.*, 2019).

It is also possible to verify that the main cause of TAVB is senility, related to fibrosis of the conduction system, which can be justified by the increase in life expectancy of the Brazilian population and its consequent aging. Associated with this, a greater prescription of medications of continuous use and the possible influences and interactions of negative inotropes in the electrical pathways of the conduction system that, depending on the underlying pathology of the patient, end up resulting in the indication of implantation of implantable electronic device due to the previous comorbidity of the patient and the need to continue the use of certain medications (GOLDONI *et al.*, 2019).

In relation to smoking and chronic coronary artery disease, endothelial dysfunction is considered the first step of vascular disease. Components of cigarette smoke cause endothelial injury and dysfunction long before vascular disease events. The healthy endothelium produces vasodilating substances, including nitric oxide (NO), prostacyclin, and endothelium-derived hyperpolarizing factor. When the endothelium is injured, the synthesis and bioactivity of these vasodilators are impaired and the balance between vasodilators and vasoconstrictors is destroyed. Currently, oxidative stress and increased inflammation by cytokines, both caused by reactive oxygen in cigarette smoke, are considered in endothelial dysfunction, reducing the bioavailability capacity of NO. Inflammatory cytokines potentiate the process of atherosclerotic alteration, which leads to endothelial dysfunction. In addition, exposure to cigarette smoke results in activation, stimulation of the coagulation cascade, and impairment of anticoagulant fibrinolysis. These effects lead to the formation of progressive vascular diseases and myocardial ischemia (KONDO *et al.*, 2019).

SAH in smoking patients should be a warning sign, as smoking increases arterial stiffness that persists for a decade after cessation; this persistent arterial stiffness is also related to increased risk for cardiovascular events (JATOI *et al.*, 2007).

Diabetes mellitus is strongly associated with the presence of a third-degree atrioventricular block. This association is independent of coronary artery disease or congestive heart failure. Neuropathy and metabolic disorders, such as hyperkalemia or acidosis, may explain this association. It is believed that the occurrence of autonomic neuropathy affecting sympathetic and parasympathetic neurons in patients with DM is responsible for abnormalities in heart rate, arrhythmias and death (STEVENS *et al.*, 1998).

The prevalence of diabetic individuals requiring treatment with a permanent pacemaker indicates the susceptibility of these patients to significant bradyarrhythmia's due to changes in the typical conduction system in DM. This would also explain the increased risk of sudden death associated with DM (MOVAHED *et al.*, 2005).

In fact, bradycardia from BAVT may be a marker or associated with the risk of death, involving several mechanisms, including severe cerebral hypoflow, followed by stroke; coronary hypoflow accompanied by myocardial infarction or, in cases of bradycardias secondary to atrioventricular blocks with very slow ventricular response, the risk of potentially malignant ventricular

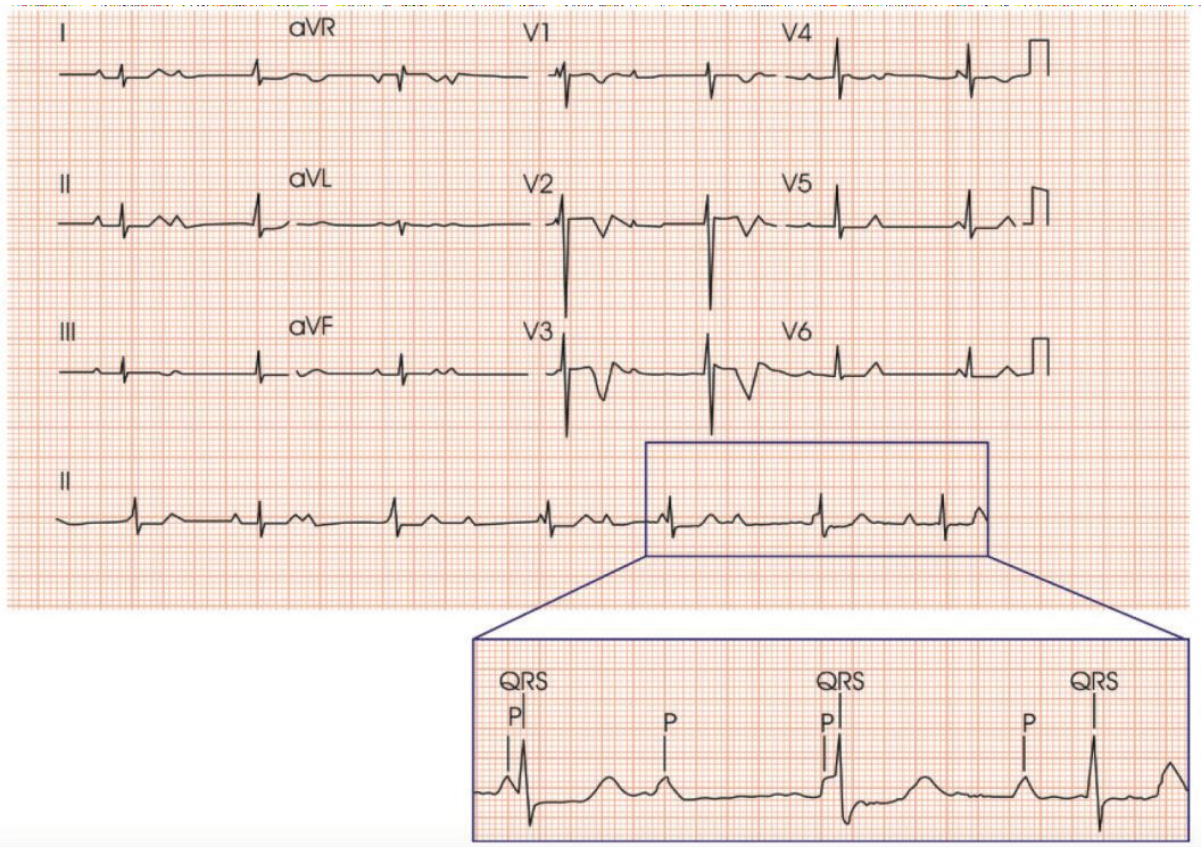

Figure 1 - Electrocardiogram with Total Atrioventricular Block.

tachycardias. The signs and symptoms, usually present in situations of low perfusion, indicate greater severity and should be the alert for prompt action in emergency rooms and intensive care (LOSCALZO, 2013).

CONCLUSION

Atrioventricular block is associated with several known cardiovascular risk factors and conditions. In this study, the risk factors are systemic arterial hypertension, diabetes mellitus, advanced age, chronic coronary artery disease and previous smoking, which together led to AV block.

Through this report and its considerations, we can observe the importance of conducting more in-depth studies in relation to BAVT in senescent patients, aiming at future publications, thus facilitating a more assertive design on the way to a correct and definitive diagnostic understanding, consequently for better care to be performed in the treatment of patients with this condition.

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