PRINZMETAL ANGINA: A CASE REPORT

ANGINA DE PRINZMETAL: UM RELATO DE CASO

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ABSTRACT

Prinzmetal's Angina (PA) is a differential and uncommon diagnosis, but not rare among other cases of chest pain. It presents with recurrent episodes of chest pain and can lead to electrocardiographic changes in different leads in each painful episode, and even with serious cardiac arrhythmias. In the literature, accurate reports on this type of variant angina are not easily found, thus making the diagnosis in most cases late and with a worse prognosis for the patient affected by the disease. Therapy, when applied correctly and as early as possible, leads to a reduction in morbidity and mortality from the disease. This work will combine a case report of PsA that occurred in the city of Valença-RJ, Brazil, correlating it with others described in the literature, to discuss and improve the diagnosis of this disease for a better prognosis for the patient.

KEYWORDS: Prinzmetal's angina; Variant angina; Chest pain.

RESUMO

A Angina de Prinzmetal (AP) é um diagnóstico diferencial e incomum, porém não raro dentre os demais casos de dor torácica. Se apresentando com episódios recorrentes de dores torácicas e podendo cursar com alterações eletrocardiográficas em diferentes derivações em cada episódio álgico, e até mesmo com arritmias cardíacas graves. Na literatura não se encontra com facilidade relatos precisos sobre este tipo de angina variante, tornando assim o diagnóstico em grande parte dos casos tardios e com pior prognóstico ao paciente acometido pela enfermidade. A terapêutica quando aplicada de maneira correta e o mais precoce possível leva a redução de uma morbimortalidade da doença. Este trabalho irá conciliar um relato de caso de AP ocorrido no município de Valença - RJ, Brazil, correlacionando com os demais descritos na literatura, afim de uma discussão e aprimoramento no diagnóstico de tal doença para melhor prognóstico ao paciente.

PALAVRAS-CHAVE: Angina de Prinzmetal; Angina variante; Dor torácica.

1. INTRODUCTION

Prinzmetal's angina (PA) is considered a differential diagnosis of chest pain. The cause is not yet well defined, according to some authors[1] it occurs due to "hypercontractility of vascular smooth muscle due to vasoconstrictor mitogens, leukotrienes or serotonin". It is caused by focal spasm of an epicardial artery, which can occur in both healthy vessels and vessels with atherosclerotic lesions. Angina due to vasospasm is uncommon, representing 2% of unstable angina investigated by coronary angiography[2]. However, the prognosis of the disease depends on the presence and extent of fixed atherosclerotic lesions[3].

The only risk factor documented to date is smoking, however, in a literature review, there is a greater number of reports of PA cases in male patients. It is five times more prevalent in men[4]. And the age of patients who suffer from the variant form of angina tends to be lower than that of patients who have coronary disease[2,4].

It presents with recurrent manifestations of chest pain, generally at rest, more common at night or early in the morning, unrelated to physical exercise, presenting transient elevations in the ST segment evidenced on an electrocardiogram. Changes in the ST segment can be present in any derivation. This same change disappears after pain regression[5].

For treatment, the use of calcium channel antagonists has level of evidence B, and together with the use of nitrates they are the best forms of treatment. The use of nitrates, both oral and intravenous, is associated with the cessation of precordial pain, while the use of calcium antagonists is related to a decrease in the number of recurrences. Since, according to the literature[6], calcium channel antagonists prevent the flow of calcium into the intracellular space, including cardiac muscle cells, conduction cells and vascular smooth muscles, by competitive blockade. Reducing excitability and heart rate, and with prolonged periods of relaxation. Leading to a decrease in oxygen consumption, afterload, contractility, and heart rate alongside an improvement in oxygen supply through the coronary vasodilator effect. The use of nitrates is not related to a reduction in mortality; however, its venous dilatation effect will reduce venous return to the heart and the end-diastolic volume of the left ventricle, reducing oxygen consumption by the myocardium[3,6].

Furthermore, it has a vasodilatory effect on normal coronary arteries or those with atherosclerotic plaques, increasing coronary collateral circulation and inhibiting platelet aggregation. In patients with associated coronary obstructions, revascularization is necessary[7].

According to a study[3,] "patients usually have a good prognosis, with the development of ventricular arrhythmias or sudden death being very infrequent". According to another author[8], attacks almost always end spontaneously, but if they continue for a long time, they can lead to death.

One author[9] concluded that patients with variant angina with severe arrhythmias during spontaneous attacks differ from other patients with variant angina only in the degree of ischemia during attacks, as reflected in maximum ST elevation, but are at a much greater risk of sudden death.

The objective of this work is to report a case of PA that occurred at the Hospital Escola de Valença-RJ, Brazil, of the Faculty of Medicine of Valença-RJ, correlating clinical data and management with others found in the literature.

This is an observational and qualitative study, reporting a case that occurred at HELGJ, Valença, Brazil, based on a bibliographic review for the theoretical foundation of the article. Articles published between 2009 and December 2019 were selected in the PubMed, Scielo and UpToDate databases, with the following keywords: Prinzmetal angina, variant angina, and chest pain.

Data collection was carried out by observing a patient with the disease during his hospitalization at the Hospital Escola de Valença/RJ, Brazil, in 2019. The instrument used for this collection were the medical records and evolutions during the patient's hospitalization. The discussion is based on the patient's step-by-step workup based on the author's view of the etiology, diagnosis, and treatment of PA.

After obtaining data collection, they were analyzed according to their thematic content and organized into systematic material, being classified, according to the information by categories, in the registration unit. The analysis was processed in three phases: in the first phase, the material was organized, and the ideas were systematized; in the second phase, a classification of information into categories, in a records unit and the third phase refers to the treatment and interpretation of data based on literature.

Complying with the ethical precepts of Resolution 196/96 for research with human beings, the project was sent and approved by the Ethics and Research Committee of the Faculty of Medicine of Valença, Brazil

2. CASE REPORT

Patient male, 60 years old, black, resident in the district of Quirino – Valença/RJ, bar owner, smoker for around 40 years/pack, without other related comorbidities, was admitted to the Hospital Escola de Valença, after episodes recurrent chest pain, in the precordium, of moderate intensity, without irradiation, intermittent, generally starting at rest. Upon admission, he was asymptomatic, showed positive myocardial necrosis markers (quantitative troponin 72 ng/L), and ECG with a plus minus pattern in precordial leads, reminiscent of the pattern described in Wellens Syndrome (characterized by the following clinical and electrocardiographic criteria: 1) waves Biphasic or deeply inverted T in V2 and V3 or. occasional. V1. V4. V5 and V6; 2) normal or minimally elevated cardiac enzymes; 3) normal or minimally elevated ST segment (< 1 mm); 4) no loss of R wave progression in precordial leads; 5) absence of pathological Q waves; and 6) anginal chest pain[10,11].

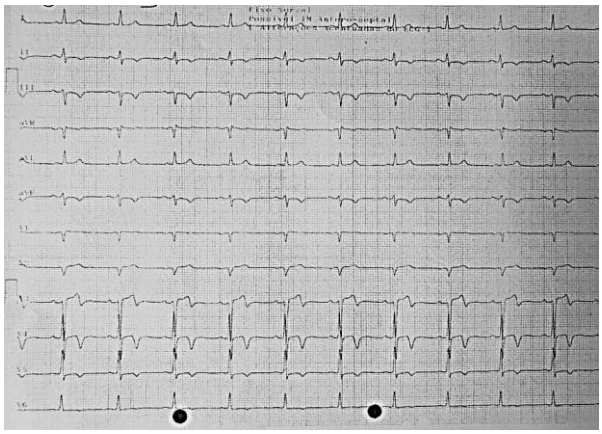

Figure 1. Electrocardiogram on admission.

He was sent to the Intensive Care Unit where after 24 hours he presented a new condition of chest pain, of moderate intensity, without irradiation, and intense sweating. A new ECG was performed which showed ST segment elevation in the anterior wall. He opted for thrombolysis with Alteplase, in addition to analgesia measures and requested coronary angiography. The patient presented coronary reperfusion criteria with cessation of pain and reduction of ST segment elevation on electrocardiogram.

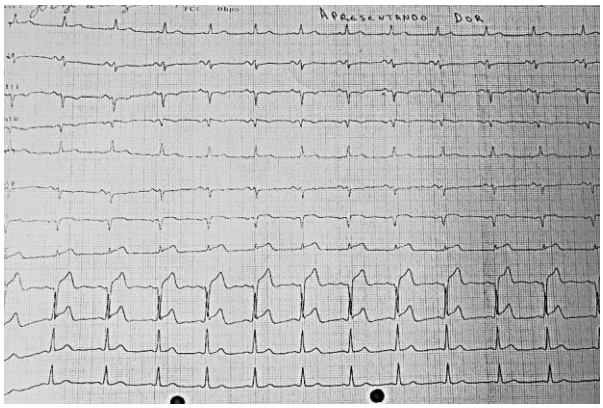

Figure 2. First episode of pain after admission.

Echocardiogram (ECG) showing mild left ventricular concentric hypertrophy, good global and segmental systolic function, grade I left ventricular diastolic dysfunction, ejection fraction 60%.

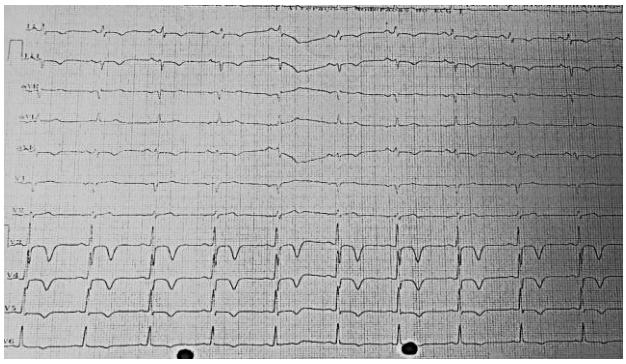

Figure 3. Electrocardiogram after thrombolysis.

The patient remained hemodynamically stable and without new episodes of chest pain, being transferred to the Internal Medicine ward after 48 hours.

After 48 hours in the ward, while waiting for a place to undergo coronary angiography, the patient presented a new episode of severe chest pain, with cold sweating, and was taken back to the ICU. A new ECG showed ST segment elevation in the inferior walls and right ventricle. A new thrombolysis was chosen, however, after 15 minutes of infusion, the patient developed a reduced level of consciousness and pulseless ventricular fibrillation. After thrombolysis was interrupted and cardiopulmonary resuscitation and defibrillation were initiated, the patient returned to sinus rhythm. A head tomography was performed without evidence of bleeding.

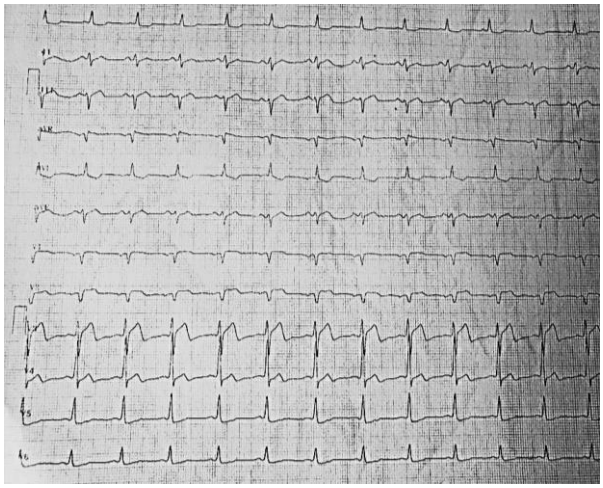

Figure 4. Electrocardiogram after pain in the ward.

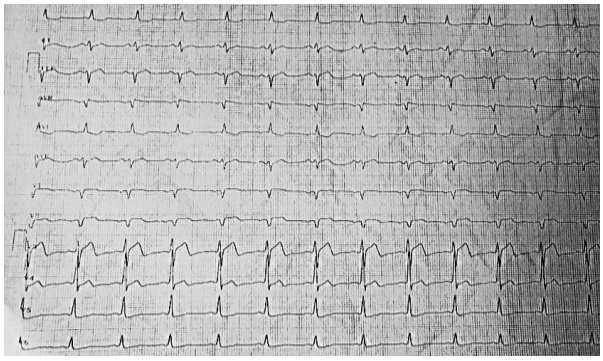

Figure 5. Post-defibrillation electrocardiogram.

New echocardiogram with cardiac chambers with normal dimensions, good global and segmental systolic function of the left ventricle, concentric hypertrophy of the left ventricle of mild degree, diastolic dysfunction of the left ventricle grade I, ejection fraction 62%.

Coronary angiography was performed with arteries free of significant atheromatosis and the presence of an important intramyocardial path in the anterior descending branch.

Therapy was started with Isosorbide Mononitrate 20 mg every 12 hours and Diltiazem 30 mg every 8 hours, subsequently Diltiazem 60 mg every 8 hours. The patient had satisfactory heart rate and blood pressure control, without new episodes of chest pain, and was discharged from hospital and outpatient follow-up with the cardiology service.

Echocardiogram 15 days after the event: slight left ventricular concentric hypertrophy, grade I diastolic dysfunction, mild mitral insufficiency, ejection fraction 64%.

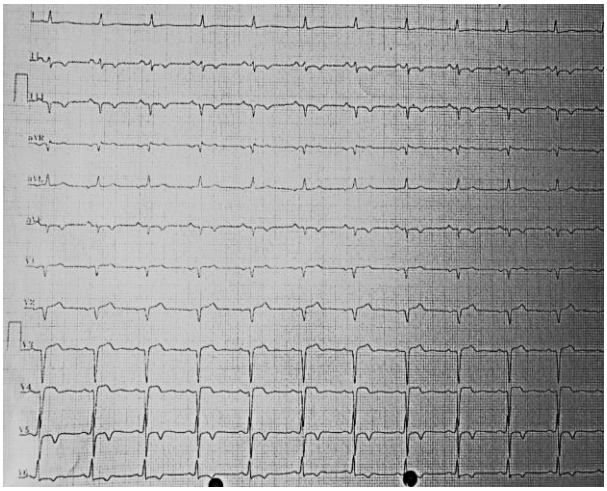

Figure 6. Electrocardiogram at hospital discharge.

This patient continues to be monitored at the cardiology outpatient clinic, after being instructed on the need to correctly use medications and the risks involving his pathology.

3. DISCUSSION

Angina due to vasospasm, PA, affects approximately 2% of cases of chest pain that are referred for investigation by coronary angiography[2]. Marked by recurrent episodes of chest pain without triggering factors and with spontaneous improvement in most cases. This fact causes the patient to delay seeking care in most cases, as in this report[5,6].

Upon admission to emergency care, the patient may present with typical chest pain, relieved using nitrates and electrocardiographic changes compatible with acute myocardial infarction, being managed as such, and measures such as thrombolysis being carried out, but will not respond to such measures, as Prizmetal's Angina is caused by a focal spasm of an epicardial artery and not a fixed obstruction. Even though the prognosis is strongly related to the presence and fixed

extent of atherosclerotic plaques[3].

Coronary angiography without significant changes together with recurrent episodes of chest pain, and in some cases with electrocardiographic changes in different leads, should make us reflect and look for different causes for the case. Appropriate therapy promotes improvement in patients' symptoms. Delays in correct diagnosis delay the start of treatment, increasing the chances of unfavorable outcomes, even leading to the risk of sudden death[12].

Patients who present variant with severe arrhythmia differ from other cases of variant angina due to the degree of ischemia and the chance of sudden death[9]. The patient reported in the case presented such arrhythmia, presented pulseless ventricular fibrillation, requiring cardiopulmonary resuscitation and defibrillation maneuvers, however, he presented a favorable outcome[2,7].

According to data analyzed in databases, the patient with the reported case had the only documented risk factor for variant Angina, smoking. Furthermore, according to the literature[4], the condition is five times more common in men, and generally, in younger patients compared to patients with coronary artery disease, the reported patient is in the age range of the other cases reported in the literature.

After starting ideal therapy for the diagnosis of variant Angina, with the use of nitrate to stop chest pain and non-dihydropyridine calcium channel blockers to avoid new episodes, controlling heart rate and blood pressure levels, according to medical data[6,] maintaining a longer period of relaxation in cardiac muscle cells and vascular smooth muscles, the patient stopped having episodes of chest pain and visits to the emergency room. Nitrates, even though they do not improve the mortality of the disease, have a venous dilatative effect, reducing venous return, and a coronary vasodilator, thus increasing coronary collateral circulation, promoting symptom relief, as described in the Brazilian cardiology guideline on unstable angina and acute heart attack. myocardium without ST elevation. As described in the literature, the patient's condition improved. Presenting an improvement in their quality of life and reducing the chances of experiencing a new episode of serious arrhythmia and sudden death[8,12.]

4. CONCLUSION

Case reports like this are necessary to better understand the different causes of chest pain, its various presentations, diagnostic and therapeutic approaches. Even though it is not a common presentation of chest pain, affecting around 2% of cases, PA is present in emergency rooms, and is most often under-diagnosed or treated incorrectly. This delay in diagnosis and initiating correct treatment increases the chances of a negative outcome for the patient, which can lead to serious cardiac arrhythmias and even sudden death.

A good anamnesis, to identify the pattern and type of chest pain, in addition to serial electrocardiograms and serial myocardial necrosis markers, help in differentiating a condition from coronary artery disease. However, in some cases, only in the imaging study through coronary angiography such differentiation is perceived.

A search in the literature and frequent updates, in addition to a greater number of reported cases, lead to greater knowledge about this pathology, which can be applied in medical practice to benefit the patient, reducing morbidity and mortality.

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