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TAKOTSUBO SYNDROME AND COVID-19: A CASE REPORT

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ABSTRACT

COVID-19 is an emerging disease, first identified in Wuhan, China and which acquired pandemic status in early 2020 according to WHO. Clinically, it presents with cough, fever, odynophagia, runny nose, prostration, abdominal pain and progressive dyspnea. However, far from causing only a disease of the respiratory system, infection by the virus causes widespread inflammation, which can cause severe complications, demonstrating varying levels of severity for those affected by this pathology. Among the most important complications, those associated with the respiratory and cardiovascular system stand out, such as severe pneumonia with pulmonary fibrosis, endocarditis, heart failure, venous and arterial thromboembolism and pulmonary hypertension with pulmonary edema. Takotsubo Syndrome (TS), also known as broken heart syndrome, is characterized by acute left ventricular failure, with dysfunction of the leftventricle wall whose causes can be idiopathic, due to physical or emotional stress. The aim of this study was to report the occurrence of TS in a patient admitted to an intensive care unit in a city in the interior of the state of São Paulo (SP), diagnosed with COVID-19 by RT-PCR. The methodology consisted of reviewing the patient's medical record, as well as diagnostic tests performed during hospitalization, associating a study of the literature on the two pathologies. Case report. A 75-year-old female patient, complaining of cough, fever of 38 °C and progressive dyspnea, was admitted to the emergency room and admitted to the ICU, with a positive RT-PCR COVID-19 test, progressing to sustained ventricular tachycardia. After reversing the tachyarrhythmia, she showed signs of acute myocardial infarction with ST-segment elevation (AMI with STSE) , but on echocardiogram, medium-apical hypokinesia of all walls and an ejection fraction (EF) of 32%. Emergency cineangio corona riography did not show significant obstructive lesions and ventriculography showing apical swelling. Early recognition of TS is essential for the accurate management of complications and for reducing morbidity and mortality in patients with cardiovascular repercussions in the presence of viral infections, such as COVID-19.

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INTRODUCTION

At the end of 2019, in Wuhan province, China, a mutagenic virus from the coronavirus family was identified in patients admitted to local hospitals who had symptoms of atypical pneumonia, which quickly progressed to Severe Acute Respiratory Syndrome. At the beginning of January 2020, this new virus was named, according to the World Health Organization (WHO) as SARS-CoV-2, and the disease that became a pandemic and has already caused more than 800 thousand fatal victims and about 25 million infected, was named COVID-19 (2019 Coronavirus Disease) (HARAPAN *et al*., 2020) (WANG *et al*., 2020) (SOHRABI *et al*., 2020) (DHAMA *et al*., 2020) (OMS, 2020). Infection with the new coronavirus can develop a respiratory disease whose progression can be mild and benign, and even severe complications that can lead to death. In mild and moderate forms, COVID-19 can present with cough, anosmia, runny nose, odynophagia, fever, prostration, abdominal pain and mild dyspnea. Severe forms, however, are varied and cause extensive viral pneumonia, with inflammation of the pulmonary parenchyma that

radiologically presents a ground-glass pattern (García, 2020) (Velavan e Meyer, 2020) (Maggi *et al*., 2020) (ALI e Alharbi, 2020) (Lauxmann *et al*., 2020). Among the main risk factors for severe disease progression are advanced age, immunosuppression and chronic diseases of the cardiovascular system, such as obesity, high blood pressure, diabetes and metabolic syndrome. Regarding complications, we can highlight the Acute Respiratory Distress Syndrome, renal failure, pulmonary thromboembolism and diseases of the cardiovascular system, such as infectious myocarditis, cardiotoxicity with myocardial injury, acute coronary syndrome and heart failure (LI *et al*., 2020) (Matias *et al*., 2020) (Bikdeli *et al*., 2020) (Kochi *et al*., 2020) (Tomasoni *et al*., 2020) . Takotsubo syndrome (ST), also known as broken heart syndrome, is characterized by acute left ventricular failure, with dysfunction of the left ventricle wall whose causes can be idiopathic, due to physical or emotional stress. The clinic is similar to that of acute myocardial infarction, with chest pain, dyspnea and syncope, which can be confused with this coronary event (CURRAGH *et al*., 2020) (KHALID *et al*., 2019).

Pathophysiology of TS is not yet fully understood. Though, it is known that in patients who suffer from this condition, there is a close relationship between the nervous system and the heart, with a hugerelease of catecholamines, which hyperstimulate myocardial β-2 adrenergic receptors. However, another important association found in the occurrence of TS is in relation to the female sex, this population is more affected when compared to men, in a proportion of 9: 1. The diagnosis is based on evidence of dyskinesia or akinesia of the ventricular wall, exclusion of acute coronary event, myocarditis and pheochromocytoma, and presence of changes on the electrocardiogram (ECG) , and echocardiogram (Moscatelli *et al*., 2019) (Napp e Bauersachs, 2020) (Amin *et al*., 2020) (Pasupula *et al*., 2019) (Boyd e Solh, 2020) . The occurrence of Takotsubo syndrome in patients who developed COVID-19 has been reported in some studies, demonstrating a possible relationship between these two pathologies. Consequently, it is important to establish the determinants for its presentation to better elucidate its consequences and complications in these patients (COSTA *et al*., 2020) (LONG *et al*., 2020) (GIUSTINO *et al*., 2020) (ROCA *et al*., 2020) .

Objectives

To report a case of Takotsubo Syndrome in a patient admitted by COVID-19 to an intensive care unit of a hospital in the city of Marília, in the state of São Paulo, Brazil, correlating with the available literature.

METHODOLOGY

The information contained in this report was obtained by reviewing the medical records of the inpatient, as well as evaluating the conducts performed, laboratory and imaging tests requested and other diagnostic methods, with a review of the available literature.

Case Description: A 75-year-old female patient started with a runny nose and fever of 38 °C three days after admission. She reported onset of dyspnea on small efforts and sought emergency services. She presented hypoxemia at admission, with oxygen saturation 88% and respiratory rate of 24 incursions per minute. She was admitted to the intensive care unit for supplemental oxygen and respiratory surveillance. As a personal history, she reported type II diabetes, systemic arterial hypertension and grade II obesity. RT-PCR COVID-19 test was performed, with positive results and computed tomography of the chest evidencing approximately 30% peripheral ground glass and in posterior lobes. On the second day of hospitalization, she presented sustained ventricular tachycardia, without signs of hemodynamic instability, and opted for chemical reversion with 150 mg Amiodarone.

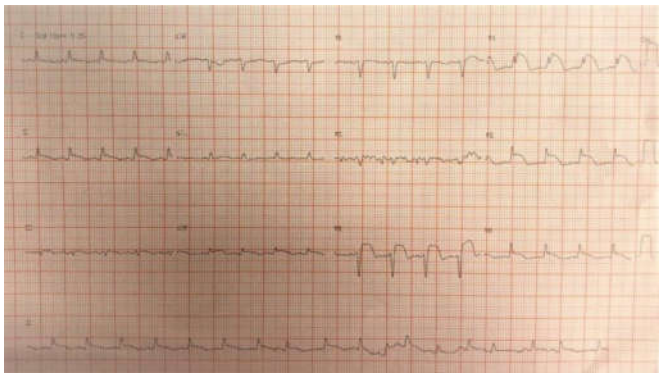

Figure 1. Electrocardiogram at rest showing TS-segment elevation in leads V3 to V6 and DII and aVF

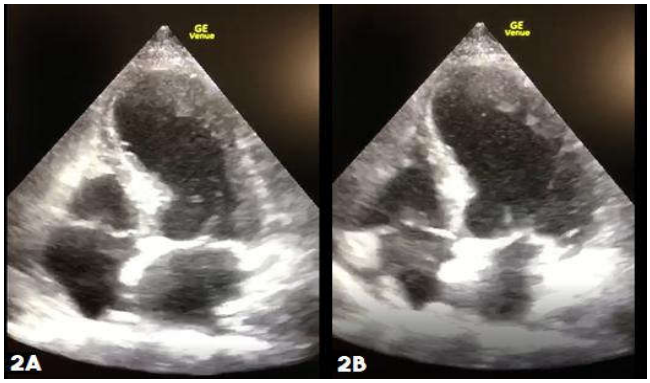

Figure 2A. Echocardiagram: apical section with 4 chambers in systole, showing middle-apical hypokinesis of all the walls of the left ventricle and middle apical swelling. Figure 2B - Echocardiagram: apical section with 4 chambers in diastole.

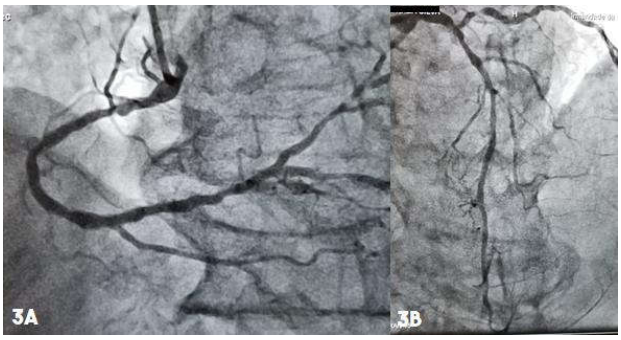

Figure 3A. Cineangiocoronariography showing right coronary artery without significant stenosis. Figure 3B - Left anterior oblique cineangiocoronariography with no evidence of significant stenosis in the anterior descending artery or circumflex artery

Figure 4. Ventriculography showing middle apical swelling

After tachyarrhythmia reversal, an electrocardiogram (Figure 1) was carried out showing signs of ST segment elevation acute myocardial infarction (AMI with STSE) . Immediately, she was transferred to the hemodynamics laboratory, with the request for emergency coronary angiography. Echocardiogram (Figure 2A and 2B), performed at the bedside, showed middle-apical hypokinesis of all walls and Ejection Fraction (EF) of 32% (using the Simpson method). Emergency cineangiocoronariography did not show significant obstructive lesions (Figure 3A and 3B). Additionally, ventriculography was performed showing apical swelling (Figure 4). After 10 days of hospitalization, patient was discharged with optimized drug therapy for ventricular dysfunction.

DISCUSSION

Takotsubo cardiomyopathy and COVID-19: possible mechanisms: TS is characterized by acute regional left ventricular (LV) systolic dysfunction mimicking acute coronary syndrome. The diagnosis of this syndrome is made when the patient presents the four Mayo criteria, namely: transient regional LV dysfunction, ST segment elevation and T-wave inversion on ECG, or troponin T elevation, absence of acute coronary obstruction and absence of myocarditis or pheochromocytoma (Sattar *et al*., 2020) (Taza *et al*., 2020) (Roca *et al*., 2020) (Giustino *et al*., 2020) (Mahajani e Suratkal, 2016) (Meyer *et al*., 2020). Pathophysiology is based on vasospasm of the coronary artery, leading to left ventricular dysfunction by an ischemic mechanism, caused by a catecholaminergic discharge secondary to intense sympathetic activation. In fact, this syndrome has a strong emotional factor, however, it is also associated with direct cardiac injuries and systemic changes, such as respiratory failure and infections. Most patients who develop TS have a benign course in evolution, recovering in 2 months, however, complications can arise, such as severe left ventricular dysfunction with apical swelling, acute heart failure, cardiogenic shock, obstruction of the LV outflow tract, formation of thrombi and arrhythmias (Faqihi *et al*., 2020) (Minhas *et al*., 2020) (Tsao *et al*., 2020) (Pasqualetto *et al*., 2020) (Giustino *et al*., 2020). Reports in the literature have already described cases of cardiac dysfunction associated with viral infections, such as Parvovirus B19 and Cytomegalovirus. The virus can act on the heart directly or indirectly. In direct injury, by degrading cardiomyocytes, the virus leads to myocarditis, heart failure and dilated cardiomyopathy.

In indirect injury, endothelial dysfunction, inflammation, activation of thrombosis and vasospasm occur, which leads to regional cardiac dysfunction due to ischemia. Cases of arrhythmia and pericarditis associated with viral infections have also been reported (Roca *et al*., 2020) (Minhas *et al*., 2020) (Long *et al*., 2020). The probable mechanisms of cardiac changes in the course of COVID-19 include vascular dysfunction, vasoconstriction and direct cardiac injury by the virus, due to the significant presence of ACE2 (angiotensin-converting enzyme 2) in the cardiomyocytes and endothelium, which constitutes the main ligand of SARS- CoV-2. In addition, the adrenergic discharge caused by the inflammatory storm in the presence of COVID-19, resulting from the intense activation of lymphocytes, could influence cardiac function, leading to exacerbations of underlying cardiopathies or acute cardiogenic shock, being the mechanism most likely involved in the development of TS in the COVID-19 course (Roca *et al*., 2020) (Minhas *et al*., 2020) (Tsao *et al*., 2020). In addition, the pro-coagulant state, caused by high systemic inflammatory activity, could contribute to acute vascular events in COVID-19. The high release of cytokines, such as IL-6, and other inflammatory and thrombotic biomarkers, such as ferritin and D-dimer, were frequent findings in patients diagnosed with COVID-19 with later development of TS. In addition to TS, cases of Reverse Takotsubo Cardiomyopathy associated with COVID-19 have also been reported, a rare variant characterized by basal and medial ventricular akinesia, without apical changes (FAQIHI *et al*., 2020) (SALA *et al*., 2020) (ROCA *et al*., 2020) (MINHAS *et al*., 2020) (TSAO *et al*., 2020).

Takotsubo cardiomyopathy and COVID-19: case reports: The case reported in the present study shows a classic history of SARS-CoV-2 infection in a 75-year-old female patient, such as respiratory symptoms and ground-glass patterns on computed tomography. During evolution, ventricular tachycardia started and what was believed to be an acute coronary syndrome on ECG, with classic signs of this syndrome such as ST segment elevation. Despite this, no coronary obstruction was found and the echocardiogram revealed diffuse hypokinesia with decreased systolic function, in addition to apical ventricular swelling. All of these data refer to the diagnosis of TS. Similar patterns were found in other case reports involving the diagnosis of ST in the course of COVID-19, including risk factors such as age and comorbidities. The study by Giustino, Croft (GIUSTINO *et al*., 2020) demonstrated that approximately 4.2% patients with COVID-19, undergoing a transthoracic echocardiogram for some reason, presented alterations compatible with TS, such as hypokinesia or regional akinesia and reduced left ventricular systolic function. On the electrocardiogram, changes such as diffuse ST elevations and T-wave inversion were also reported. These patients also demonstrated high levels of troponin I and CK-MB. Despite these findings, patients with TS showed the lowest levels of inflammatory and prothrombotic biomarkers. Finally, the presence of TS was associated with higher rates of unfavorable outcome in the course of COVID-19 when compared to patients without TS.

In the case report of Roca, Lombardi (ROCA *et al*., 2020) , an 87-year-old woman with a history of breast cancer presented with symptoms of COVID-19 and evolved with tachycardia and increased troponin I and CK- MB on the second day of hospitalization. An electrocardiogram was carried out, which showed negative T waves. Echocardiogram showed apical swelling and hypokinesia, with reduced systolic function, with a final diagnosis of TS. The treatment based on this diagnosis consisted of Bisoprolol and Fondaparinux, which progressed to hospital discharge without further complications. Minhas *et al*. (MINHAS *et al*., 2020) reported a case of a 58-year-old woman with a history of type II diabetes mellitus, hypertension and dyslipidemia, who initially presented with respiratory symptoms and progressed to respiratory failure, in addition to sinus tachycardia, ST segment elevation, diffuse PR interval depression and changes in the T wave and ST segment on ECG. There was also an increase in serum troponin I levels. PCR test for COVID-19 returned positive, confirming the diagnosis. Echocardiographic investigation showed akinetic and hypokinetic areas, associated with apical swelling and reduced left ventricular systolic function, classic findings of TS. Thereafter, dual antiplatelet therapy and anticoagulation with continuous intravenous heparin was started. For COVID-19, hydroxychloroquine and azithromycin were started, but the first was discontinued after echocardiographic findings. The patient evolved with cardiogenic shock, requiring dobutamine, however, the ventricular function was recovered after a few days, which corroborates the hypothesis of a pathological cardiac mechanism resulting from stress as a result of COVID-19. Finally, it is important to note that several studies have warned about the risk of emergency coronary angiography in patients with COVID-19. This procedure has been delayed in several cases reported after positive testing for COVID-19, due to the risk of viral spread to both the patient and the examiner (PASQUALETTO *et al*., 2020) (MINHAS *et al*., 2020) .

FINAL CONSIDERATIONS

Taking into account the case report presented and the discussion, we conclude that TS should be considered as a differential diagnosis in patients with COVID-19 and Acute Coronary Syndrome with ST segment elevation. Furthermore, the opposite is also valid, that is, COVID-19 should be considered as a diagnosis in patients who present with cardiac complications corresponding to TS during the pandemic. The exact mechanism by which the virus leads to cardiac changes remains unknown, however, it is possibly related to stress-induced cardiomyopathy, due to the high release of pro-inflammatory cytokines and catecholamines and due to the high inflammatory activity of lymphocytes during infection. However, we cannot exclude the possibility of direct cardiac injury, due to the tropism of

SARS-CoV-2 by the enzyme ACE2, which is also expressed in cardiomyocytes and endothelium. Finally, we conclude that the investigation with echocardiogram should not be delayed in patients with alterations suggesting ischemia on the electrocardiogram, due to the importance of this exam for the correct diagnosis of TSEarly recognition of the syndrome is essential for the precise management of this complication and for reducing morbidity and mortality in patients with cardiovascular complications in the presence of viral infections, such as COVID-19.

Conflicts of Interest: The authors declare no conflicts of interest.

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