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# MYOCARDIAL INJURY IN PATIENT WITH COVID-19 INFECTION: EXPERIENCE OF A CASE


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| **Nataly Sofía Valdiviezo Allauca** | General Practitioner/Riobamba-Ecuador *Corresponding Author |
| **Mayra Alexandra Yauli Santos** | Medical Resident at "Hospital Básico Baños"/Ambato-Ecuador |
| **Lineth Carolina Zurita Mayorga** | Medical Resident at "Hospital General Docente Ambato"/Ambato-Ecuador |
| **Fanny Gabriela Barragán Quishpe** | General Practitioner at "Centro de Salud Chimbo"/Guaranda-Ecuador |
| **Diana Alexandra Cóndor Muñoz** | General Practitioner at "Puesto de Salud Oyacachi"/ Yaruqui-Ecuador |



**ABSTRACT** The World Health Organization (WHO) on December 31, 2019, was notified by China in the city of Wuhan of several cases of pneumonia of unknown cause. Thus, thanks to the samples obtained from infected people, the existence of a new coronavirus is reported. I call it SARS-CoV-2 and the disease COVID-19; but its rapid spread has led to it being declared a pandemic. The cardiovascular manifestations of COVID-19 infection are still uncertain, the existing literature is scarce. 1

However, several cases of cardiovascular disease have been published, which is a marker of poor prognosis. Acute cardiac injury, defined as significant troponin elevation, is the most commonly reported cardiac abnormality in COVID-19 infection.2

Reason why we present the case of an octogenarian patient, with multiple comorbidities; who developed acute coronary syndrome, shock of mixed etiology and dyspnea in the context of COVID-19 infection. Complications were mainly related to pneumonia and acute respiratory distress syndrome that led to his death.

**Objective:** Describe the myocardial injury that Covid-19 infection produces in patients with cardiovascular risk factors.

**Methodology:** This is a systematic review of Covid-19 infection and cardiological injury, emphasizing its clinical, laboratory and radiological characteristics. The information and images obtained belong to the medical personnel in charge of the case, whose reinforcements rest on the statistical package Excel, Word and JPG.

**Conclusion:** Most of the information available on cardiac involvement by coronavirus comes from reports of hospitalized patients in Wuhan; who investigated cardiovascular complications based on clinical detection and diagnostic studies. It is for this reason that this case of acute coronary syndrome is presented in a patient with COVID-19 infection, in which his predisposition to suffer a rapid onset shock stands out.


**KEYWORDS :** Myocardial Injury, Covid-19, Cardiovascular Risk

## INTRODUCTION

Coronavirus is an enveloped single-stranded RNA virus; 2/3 of its genetic material translate to 16 non-structural proteins and 1/3: 4 structural proteins: protein S, with the S1 subunit which intervenes in the affinity of the virus for the angiotensin-converting enzyme 2 (ECA2); the S2 subunit, which facilitates the fusion of the cell membrane; the M protein, which allows the release of the RNA to the host cell; and proteins N and E, which are structural proteins responsible for interacting with the host's innate immunity.[3]

During the initial stages of the pandemic, and given the few published studies on this, this coronavirus was believed to cause purely respiratory symptoms; however, as the number of patients increased, cardiovascular disease was found to play a critical role in the development and prognosis of infection. ACE is an enzyme that helps convert angiotensin I to angiotensin II, it is located at the lung level and in the vascular endothelium predominantly, but also in other tissues.[4]

Recent bibliography emphasizes that the administration of ACE inhibitors (ACEI) or angiotensin II receptor blockers (ARAII) favored a greater expression of ACE and ACE2; the latter is in charge of converting angiotensin II into angiotensin I-7 (Ag1-7) and angiotensin I into angiotensin 1-9 (Ag1-9), which have vasodilatory, antiproliferative and natriuretic effects contrary to angiotensin II. Together, ECA2 is considered the entry site for SARS-CoV-2 into the cell.[5,6]

With all that said and thanks to the information from experimental studies, it could be suggested that patients with chronic diseases who are treated with ACE inhibitors or ARA II, may be susceptible to a greater impact of COVID-19 infection, this associated with overexpression of ECA2. Furthermore, the inflammatory response and associated hemodynamic changes are the probable triggers for the increase in the incidence of acute myocardial infarction. Some of these cases may present with overlapping symptoms. [7]

Chest pain and typical electrocardiographic changes have been reported in coronary artery patients with significant lesions, which were positive for COVID-19. This would explain the increase in mortality documented in cardiac patients infected with this virus. [8]

## CASE PRESENTATION

This is an 81-year-old male patient, resident in Pichincha, Ecuador, by profession a professional driver, with a history of type 2 diabetes mellitus on insulin NPH treatment, high blood pressure on Enalapril treatment, dyslipidemia on simvastatin treatment, and ischemic stroke in 2016, all of

them controlled.

The patient presented with symptoms of generalized discomfort, dry cough, severe chest pain (VAS 9/10) with irradiation to the left shoulder and dyspnea of medium efforts of 12 days of evolution, with the apparent cause of having made a trip to Guayaquil, with this Symptoms come to our Teaching Hospital in Calderon-Quito.

Upon arrival at the Emergency Service, he was evaluated, finding a patient with frank respiratory failure, for which he proceeded with orotracheal intubation and invasive mechanical ventilation. Extension tests are performed: the initial electrocardiogram showed sinus tachycardia. (Photo 1)

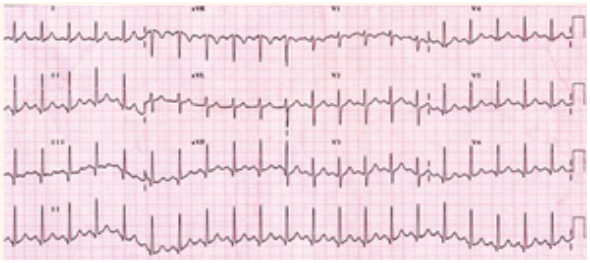

**Photo1.ECG: SinusTachycardia**

In the plain chest radiograph and tomography, the objective was: peripherally predominant cottony infiltrates in both lung fields (Photos 2 and 3).

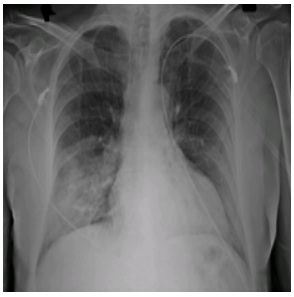

**Photo 2. Chest X-ray with peripherally predominant cottony infiltrates in both lung fields**

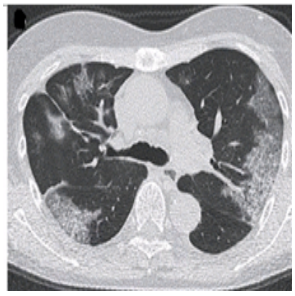

**Photo 3. Chest CT: Opacity in bibasal ground glass**

The analysis showed Leukocytes: 12,000, Neutrophils: 80.1%, Lymphocytes: 550, Hemoblobin: 14g /dl Hematocrit: 38.1%, Platelets: 332000, Creatinine 1.2 mg /dl, D-Dimer: 700ng/dl, Sodium: 131, Potassium: 4.1 , Chlorine: 92, Nasopharyngeal swab: SARS-VOC-2 PCR Positive. Troponin T: 1.14ng/ dl, creatine kinase MB at 679 U /l, cerebral natriuretic peptide: 3.06 pg /ml.

The patient evolves with hemodynamic deterioration, which culminates in shock, the same that did not revert to vasoactive support. It was decided to perform urgent cardiac catheterization, where acute thrombotic occlusion of the right coronary artery was demonstrated (photo 4); who required angioplasty, however, at the time of the procedure, the patient suffered cardiorespiratory arrest, without response to CPR maneuvers.

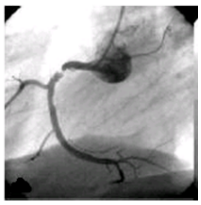

**Photo 4. Coronariography: thrombotic occlusion of the right coronaryarteryinitsmiddlesegment**

Finally, the patient died 48 hours after admission.

## DISCUSSION

Covid -19 infection and its relationship with cardiovascular pathology is still uncertain, studies have been seen where it is related to hypertensive patients receiving ACEI or AIIRA treatment. This being your increased risk of heart disease.

Increased levels of troponin, natriuretic peptides, and D-dimer have prognostic value in patients with COVID-19 infection. The literature tells us that these patients have a high risk of Myocardial Infarction (AMI), arrhythmias, heart failure, and sudden death, in relation to the systemic response to the virus and the necessary treatments in the acute phase. However, healthy individuals with severe infection can also develop cardiovascular compromise.[9,10]

Creatine kinase levels (CK-MB) are also significantly higher in patients with an ICU requirement compared to those treated in other services. These findings suggest a correlation between the severity of COVID-19 infection and the degree of myocardial involvement.[11]

Although severe hypoxemia secondary to respiratory dysfunction, by itself may explain myocardial injury, additional mechanisms are proposed. It is proposed to ECA2; This signaling pathway could have a role in the mechanisms of direct myocardial damage. Another hypothesis involves cytokine storm triggered by the imbalance between the type 1 and type 2 response of T lymphocytes.[12]

A state of hypercoagulability has been pointed out in patients affected by COVID-19, which culminates in disseminated intravascular coagulation, favoring thrombotic phenomena. Recent studies have shown that D-dimer constitutes a prognostic index of mortality.[13]

If we remember, our patient presented very high D-dimer numbers (700 ng/dl). This leads to the hypothesis that severe COVID-19 infections can act as a precipitating factor for thrombotic events, especially in patients with cardiovascular risk factors.

## CONCLUSION

Knowing about Covid-19 infection associated with cardiovascular disease, drives us to continue documenting these cases.

It is known that the infection itself or its treatment could have cardiovascular manifestations, such as heart failure, myocardial injury, ischemia, or QT prolongation. For these reasons, this clinical case was presented where we observed the impact of Covid-19 infection that led our patient to a withering scenario.

Thus, more scientific evidence and more studies on this topic are needed; Because with a greater knowledge of the clinical manifestations, in the interpretation of the different diagnostic tests and in the cardiovascular consequences of the treatments used for this disease, the chaos generated by this pandemic can be mitigated.

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