VOLUME - 9, ISSUE - 9, September - 2020 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper | Clinical Research

FULMINATING MYOCARDITIS IN PATIENT AFFECTED BY COVID-19

María Belén Paucar Valdivieso	Resident doctor at Hospital Universitario Andino Chimborazo/Ecuador
Joselyn Jennifer Castillo Jaramillo	Resident doctor at Hospital Padre Carollo Un Canto a la Vida/Quito/ Ecuador
Manuel Antonio Álvarez Pichazaca	Resident doctor at Hospital del Sur Esmeraldas/Ecuador
David Andrés Suasnavas Amagua*	General practitioner /Ecuador.*Corresponding Author

ABSTRACT

The coronavirus disease (Covid19) caused by "severe acute respiratory syndrome coronavirus 2" (SARS-CoV2), began in Wuhan, China, but its rapid spread internationally has led the World Health Organization to declare it a pandemic on March 11 last. It is essential that cardiology specialists and the medical community in general know its impact at the cardiovascular level. We must know that the information to date comes, mostly from China, from retrospective and single-center analyzes, or from reported cases. Therefore, the statistical data is probably not the real one, but it is what is available to date; and surely some of them have modifications as time goes by.1

Covid-19 infection directly impacts cardiovascular disease. Patients with heart problems are predisposed to infection by the new coronavirus, and also have a higher risk of adverse events. Thus, the infection itself is associated with complications at the heart level.2

When speaking of myocarditis, it imposes great importance on us, because it is a serious clinical entity with permanent evolution from its initial definitions at the beginning of the 19th century to the present, and it is associated with Covid-19 infection.3

Reason for which we present the case of an octogenarian patient, with multiple comorbidities; who develops fulminant myocarditis in the context of Covid-19 infection, and subsequently presented complications; among them superinfection due to severe pneumonia and acute respiratory distress syndrome that led to his death.

OBJECTIVE: Describe how myocarditis associated with Covid-19 infection causes sudden damage.

METHODOLOGY: This is a systematic review of fulminant myocarditis in the context of Covid-19 infection and its subsequent complications; emphasizing its clinical, laboratory and imaging characteristics. The information and images obtained belong to the medical personnel in charge of the case, whose reinforcements are provided by the Excel, Word and JPG statistical package.

KEYWORDS : Fulminant myocarditis, Covid-19.

INTRODUCTION

SARS-CoV2, belongs to the coronaviridae family, is a ribonucleic acid (RNA) virus. Its genome is 96.2% identical to coronaviruses found in bats. This virus can use angiotensin converting enzyme 2 (ACE2) protein to enter cells. ACE2 is highly expressed in alveolar cells of the lung, there they play roles of lung protection, which can be altered when the virus is bound to this receptor. The infectivity of Covid-19 is higher than that of influenza, with an estimated basic reproductive number (Ro) of 2.28. Their mortality is higher than that reported in the last seasonal influences (0.1%), although lower than other coronavirus outbreaks such as SARS and MERS-CoV (9.6% and 34.4% respectively), but Covid-19 had more deaths than these last, given their high infectivity and large number of affected patients. [4]

Both in advanced age, such as diabetes and dyslipidemia, there is a decrease in immune capacity, this makes patients more vulnerable to developing severe infections. Therefore, cardiac pathology is a marker of immune dysfunction and is related to the prognosis of infection. Cohort studies estimated that between 7-17% of hospitalized patients presented acute myocardial injury; it is more common in patients admitted to the intensive care unit (ICU) (22.2% vs 2.0% p <0.001) and in those who died (59% vs 1% p <0.0001) .[5]

With all that has been said, the coronavirus pandemic has generated a serious global health problem. Covid-19 mainly affects the lung, but it has been seen that in some patients, myocardial involvement also occurs, producing myocarditis and arrhythmias.[6]

Myocarditis is defined as an inflammatory disease of the heart muscle and is a major cause of acute heart failure, sudden death, and dilated cardiomyopathy. Viruses are the cause of most cases of myocarditis or inflammatory cardiomyopathy and can induce an immune response that causes inflammation despite having eliminated the pathogen. Other etiologic agents that cause myocarditis are drugs, toxic substances, or autoimmune disorders. In recent years, advances in non-invasive techniques such as cardiac magnetic resonance have been very useful to support the diagnosis of myocarditis, but toxic, infectious and inflammatory, infiltrative or autoimmune processes occur in cells, and only biopsy endomyocardial allows to establish the nature of the etiological agent. [7]

Thus, we present the case of an elderly patient, affected by multiple comorbidities, who presented Covid-19 confirmed with nasopharyngeal swab, associated with fulminant myocarditis, and after multiple complications led to an unfavorable scenario.

CASE PRESENTATION

This is an 81-year-old male patient, born and residing in Guayaquil, Ecuador, by profession a retired civil engineer; ORh positive blood group, without referring to allergies, with a history of hypertension, chronic migraine, grade II hydronephrosis, and cervical degenerative arthropathy, all of them controlled. In his usual treatment, losartan of 100mg orally every day and paracetamol 500mg orally every 8 hours stands out.

In May 2020, he visited the emergency department of the specialized hospital, with a clinical picture of 9 days of evolution of unquantified thermal rise, cough without expectoration, asthenia, and oppressive chest pain of moderate intensity without radiation; having as an apparent cause contact with a positive family member for Covid-19 for approximately 5 days. On physical examination with a blood pressure of 70/50 mmHcg, heart rate of 101 beats per minute, respiratory rate of 24 breaths per minute, temperature of 39 degrees Celsius, oxygen saturation of 85% ambient air rising to 90% with 3 liters of oxygen per nasal cannula.

Immediately proceed to hydration and medication is prescribed to improve blood pressure; Despite the fluid overload and norepinephrine, the hypotensive persisted with signs of hypoperfusion among them: skin coldness and elevated lactic acids: 3.8 mmol / l.

The electrocardiogram showed an elevation of the ST segment. (Photo 1)

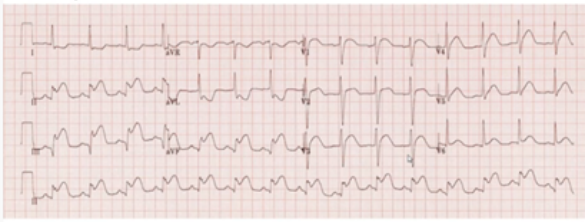

Photo 1. ECG: ST segment elevation

The polymerase chain reaction (PCR) of the nasopharyngeal swab virus was positive for SARS-CoV-2 and negative for adenovirus and Influenza A and B viruses, with a positive epidemiological environment (relatives with fever and respiratory symptoms in the previous days). Among the laboratory data, the elevation of NT-proBNP troponins 4,421 ng / l stood out, leukocytosis: 15.17 × 109 / l, lymphocytes: 3.59 × 109 / l, CRP 10mg / l, D-dimer 33,242 ng / ml .

Likewise, a simple chest tomography was performed where a typical pattern of Covid-19 is evidenced. (Photo 2-3).

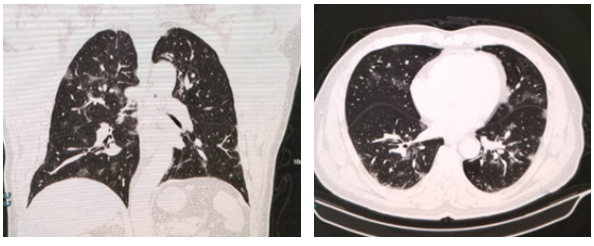

Photo 2-3. Simple chest tomography: peripherally distributed ground glass opacities in both lung fields.

Due to the highly indicative symptoms of myocarditis (diffuse ST elevation, thermal rise) and the preserved left ventricular ejection fraction, coronary angiography was not performed due to low clinical suspicion of acute coronary syndrome. In addition, clinical and chest tomography revealed bacterial superinfection, diagnosing severe pneumonia due to Covid-19; Treatment for myocarditis with corticosteroids, based on methylprenisolone: 500 mg / day in descending regimen, antibiotic treatment based on ceftriaxone and clarithromycin (2 grams every day and 500 mg every 12 hours respectively), and antiviral treatment: ritonavir 400 mg / lopinavir 100 mg / 12 h.

However, the patient presented hemodynamic deterioration with frank respiratory failure, which required cardiopulmonar y resuscitation, and high doses of vasopressors; despite this, the patient evolved unfavorably and died 72 hours after admission.

DISCUSSION

The clinical presentation of patients infected by SARS-CoV-2 is highly variable, and the respiratory symptoms are the most frequent. Given the current epidemiological situation, this etiological agent must be considered as the cause of other clinical conditions such as acute myocarditis. In series of patients hospitalized for Covid-19, it is shown that one fifth had heart damage defined by elevated ultrasensitive troponin. These patients were elderly, had more comorbidities, and had higher levels of leukocytes, C-reactive protein, and dimer D. Patients with elevated troponin had a much higher proportion of respiratory distress (58.5% vs 14.7%) and a higher mortality (51.2% vs 4.5%). [8,9]

Thus, as fulminant myocarditis is a syndrome with high morbidity and mortality, early diagnosis and correct treatment are of vital importance. In this case, a clinical picture compatible with acute myocarditis was highlighted, associated with the respiratory symptoms of covid-19, which is why he suffered complications such as bacterial superinfection, which, despite the prescribed treatment, evolved unfavorably, leading to his death.

For all that has been said, it can be said that the mechanism by which myocardial injury can be triggered is complex and multifactorial; without a doubt, the acute inflammatory response could lead to ischemia, [10,11] even more in the presence of pre-existing cardiovascular disease and bacterial superinfection such as was the exposed clinical case.

CONCLUSION

SARS-CoV-2 infection leads to cardiovascular complications or exacerbation of pre-existing heart diseases, this implies a higher risk of morbidity and mortality.[12,13]

It was seen that of the patients with Covid-19 about 7% suffer from myocarditis. Thus, when a patient with a probable diagnosis of Covid-19 is admitted to intensive care units, he already has a high index of suspicion for myocardial injury. [14]

It seems prudent in cases of fulminant myocarditis to start treatment with glucocorticoids and immunoglobulin therapy early.[15] That was the treatment that was prescribed for our patient, however, due to the bacterial superinfection, it led to a not very encouraging scenario. thus, reaching his death.

INTEREST CONFLICT

The authors of the article have no conflict of interest.

REFERENCES

1. Li B, Yang J, Zhao F et al. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China. Clin Res Cardio 2020.
2. Zheng Y-Y, Ma Y-T, Zhang J-Y and Xie X. COVID-19 and the cardiovascular system. Nat Rev Cardiol. March 5, 2020. doi: 10.1038 / s41569-020-0360-5. [epub ahead of print].
3. Ruan Q, Yang K, Wang W, Jiang L, Song J. Clinical predictors of mortality due to COVID-19 based on an analysis of data of 150 patients from Wuhan, China. Intensive Care Med 2020.
4. Zhou F, Yu T, Du R et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet 2020.
5. Elissa Driggin et al. Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the Coronavirus Disease 2019 (COVID

19) Pandemic. J Am Coll Cardiol. 2020.
6. John R Greenland, M.D, Ph D et al. COVID-19 Infection: Implications for Perioperative and Critical Care Physicians.
7. Hongde Hu, Fenglian Ma, Xin Wei, Fang. Yuan.
8. Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin.
9. Eur Heart J., (2020), http: //dx.doi.org/10.1093/eurheartj/ehaa190 | Medline. [4]
10. J.A. Fontes, N.R. Rose, D. Čiháková. The varying faces of IL-6: from cardiac protection to cardiac failure.
11. Cytokine., 74 (2015), pp. 62-68. http://dx.doi.org/10.1016/j.cyto.2014.12.024 | Medline [5].
12. WHO Statement on the third meeting of the IHR Emergency committee concerning Middle East respiratory syndrome coronavirus (MERS-CoV). Wkly Epidemiol Rec. 2013; 88: 435-436.
13. Irabien-Ortiz A, Fulminant myocarditis due to COVID-19, Revista Española de Cardiología. (2020), doi: https://doi.org/10.1016/j.recesp.2020.04.001.
14. Giudicessi JR, Noseworthy PA, Friedman PA, et al. Urgent guidance for navigating and circumventing the QTc prolonging and torsado genic potential of possible pharmacotherapies for COVID-19. Mayo Clin Proc. 2020. https://doi.org/10.1016/j.mayocp.2020.03.024.
15. Lippi G, Lavie CJ, Sanchis-Gomar F. Cardiac troponin I in patients with coronavirus disease 2019 (COVID-19): evidence from a meta-analysis. Prog Cardiovasc Dis. 2020 Mar; S0033-0620 (20) 30055-4. https://doi.org/10.1016/j.pcad.2020.03.001 PMID: 32169400.