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Myocardium Pericardial Effusion During COVID-19

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Abstract

Here we report a case of Myocardium pericardial effusion after infection with SARS-Cov-19, and the treatment with the peptide CIGB-258, CIGB-814.

***Keywords*:** *Covid-19; SARS-CoV-2; hyper-inflammation; cytokines storm; pericarditis; pericardial effusion; CIGB-258; CIGB-814*

Introduction

Reports of severe extra-pulmonary complications associated with coronavirus disease 2019 (COVID-19) may be found in the literature [1]. However, cases of myocardium pericarditis directly associated with COVID-19 have not been reported.

Materials and Methods

We describe a patient with Covid-19 developing a viral myocardium pericarditis. Academic ethics committee approval was waived because this was a single-case report; written informed consent was obtained from the patient.

Results and Discussion

A 48-years-old man without antecedent of cardiovascular diseases was confirmed as positive to severe acute respiratory syndrome coronavirus-2 (SARS-Cov-2) infection by real-time reverse-transcriptase–polymerase-chain-reaction (RT-PCR) test in May 5th 2020. On arrival, no thoracic abnormalities were noted by thorax X-ray imaging (Figure 1). Initial laboratory studies revealed hemoglobin 132 g/l, hematocrit 42 g/l, erythrosedimentation 9 mm/h, creatinine 82 mmol/L, uric acid 291 mmol/L, TGP 12 mmol/L, TGO 23 mmol/L, coagulogram TS: 1" TC: 6'. Standard treatment for SARS-Cov-2 infection, according to World Health Organization recommendations, was two tablets of Lopinavir/ritonavir (250 mg), one tablet of Chloroquine diphosphate (250 mg), every 12 hours, and 3.5 MIU of recombinant Interferon Alfa-2b (Heberon Alfa R®, CIGB, Havana), three times per week. The clinical course of this treatment was favorable.

Cardiopulmonary examination was significant for a biphasic pericardial rub, a prolonged QT interval was observed in evolution electrocardiogram (Figure 2), seven days after treatment of the infection, and echocardiogram image showed moderate pericardial effusion at anterior and posterior wall without hemodynamic consequences (Figure 3). The patient was transferred to Intensive Care Unit for close hemodynamic monitoring and special isolation with negative airflow.

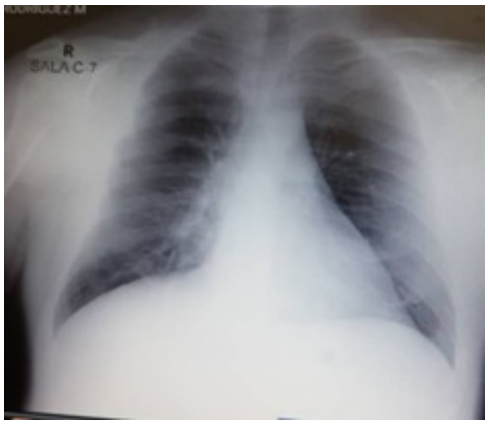

Figure 1: *Progress of thorax X-ray imaging of acute respiratory distress syndrome at patient arrival (May 5th, 2020)*

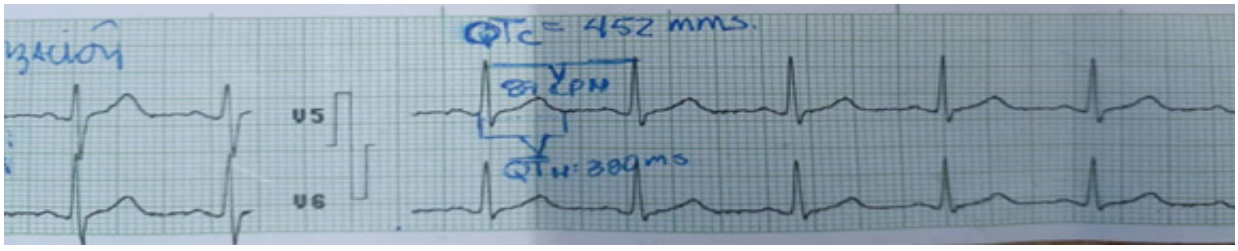

Figure 2: *Evolution electrocardiogram: Sinusal rhythm, normal QRS and PR, ST apparently normal, middle QT segment of 380 milliseconds at frequency of 87 beats per minute (QTc: 452 milliseconds calculated according Bazett's formula, interpreted as length). (May 12th, 2020)*

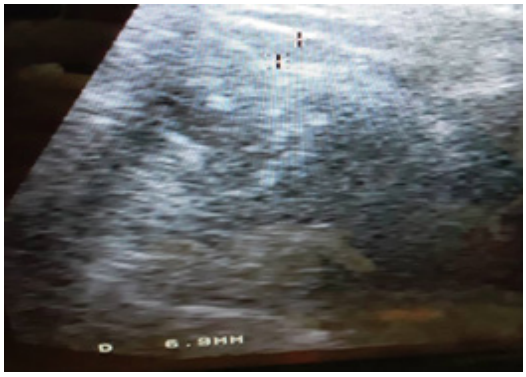

Figure 3: *Echocardiogram after seven days of Covid-19 infection showed moderate pericardial effusion*

Chloroquine diphosphate treatment was stopped on May 12th, and the patient was initiated on sodium diclofenac (75 mg) IM every 12 hours, and 1 mg of CIGB-258 (Jusvinza®, CIGB, Havana) IV every 24 hours, because this medicine seems to be effective [1] and safe [2] in COVID-19 patients under cytokine storm (Clinical trial: rpcec.sld.cu, N0 RPCEC00000313). On May 14th, the patient reported feeling much better, without deep inspiration. He was auscultated and pericardial rub was perceived in the lower left sternal border. There was evidence of a decrease in pericardial effusion. No intra-pericardial fluid was observed in the posterior and anterior wall by echocardiogram. Subsequently, treatment with Diclofenac sodium was discontinued and treatment with 60 mg of Methylprednisolone per day was initiated.

On May 16th, the patient was asymptomatic, with a pericardial effusion less than 4 mm, without friction and arrhythmias, VI 54 mm,

FEVI 59%, frequency 78 beat per minute, normal ECG, QTc 433, Leukocytes 13, Polymorphonuclear 0.94, Lymphocytes 0.05, monocytes 0.01, RT-PCR negative, and erythrocyte sedimentation 8 mm/h. After four days of treatment with CIGB-258, steroids, and Kaletra, the patient had a favorable clinical course. Thorax X-ray imaging (Figure 4), ECG (Figure 5), and ECO showed normal parameters. At sixteenth day of evolution, result TR-PCR test was negative and the patient was discharged home in stable condition.

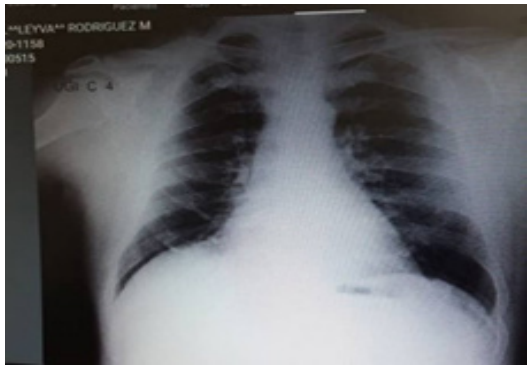

Figure 4: *Progress of thorax X-ray imaging (May 16th)*

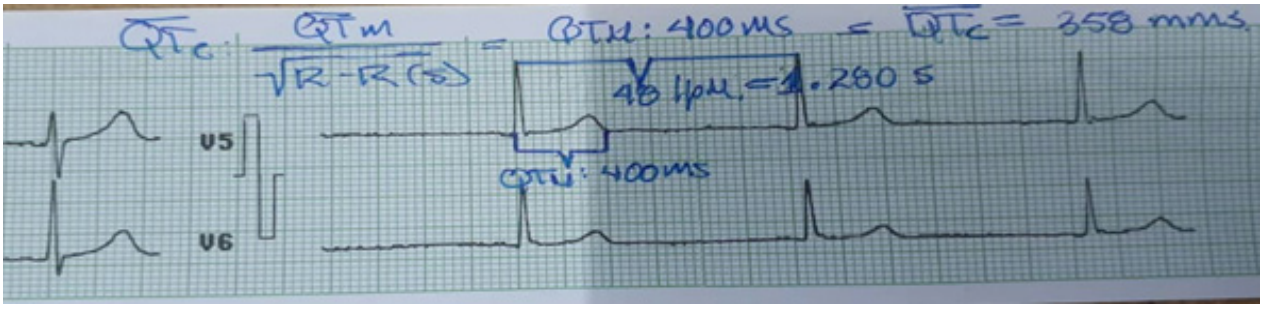

Figure 5: *Evolution electrocardiogram: Sinusal rhythm at frequency 48 beat per minute, narrow QRS, QTc: 352 milliseconds, without risk of malignan arrithmia*

Results of laboratory tests for this date were: Hemoglobin 14 g/l, Leukocytes 1010 cell/l, Polymorphs 0.80%, Lymphocytes 0.20%, Erythrosedimentation 5 mm/h, Platelets 250 mol/l, C-reactive Protein in normal values, LDH 290 IU, Triglycerides 2.3 mg/dL, Cholesterol 6.4 mg/dL, TGP 10 IU, Creatinine 81 mmol/L, GGT 20 IU, Uric acid 389 mg/dL. Extra-hospital follow-up was performed at primary health care and it was verified that the RT-PCR test performed on June 3th was negative to epidemiologically discharge the patient.

As the pandemic progress, cardiac manifestations of Covid-19 have been identified. [3,4] This patient developed a complication derived from the infection with SARS-Cov-2 known as pericardial effusion. Infections, neoplastic, autoimmune, metabolic, and drug-related causes have been reported as potential origins of this incidental finding or manifestation of a systemic or cardiac disease. [5] A second cardiovascular event converged in this patient, which required careful monitoring. ECG findings suggested adverse events caused by Chloroquine diphosphate treatment, which could be the cause of severe complications such as ventricular fibrillation and Torsaides d' Points. [6] ECG was normal 72 hour after this medicine was discontinued.

Conclusions

The medical staff concluded that an accelerated immunological response was associated to pericardial effusion, in absence of circulatory implications, and the treatment with CIGB-258, steroid, and Kaletra was prescribed. Although the mechanism of action of CIGB-258 is under investigation, this medicine, previously coded as CIGB-814, has been safe and effective inducing regulatory effects associated with the inhibition of inflammation in rheumatoid arthritis patients, without producing immunosuppression reported in

treatments with Tocilizumab, Anankira and JAK kinases inhibitors. [ ] It may not be concluded that pericardial effusion was caused by Covid-19 disease from this single-case report, but adding this to previous findings reported in the literature supports the argument in favor of this conclusion. Therefore, timely diagnosis of this cardiovascular complication is essential for making optimal therapeutic decisions. Finally, combinations of CIGB-258, steroids, and Kaletra may be a safe and effective alternative for treatment of pericardial effusion in patients with Covid-19.

Acknowledgements

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Conflict of Interest: None reported.

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