Annals of Case Reports & Reviews

Case Report

doi: 10.39127/2574-5747/ACRR:1000200
Kalkan A, et al. Annal Cas Rep Rev: ACRR-200

Cardiogenic Shock Caused by Cardiac Tamponade Due to Fulminant HHV-6 Myopericarditis In A COVID-19 Patient: A Case Report

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Citation: Kalkan A, Pennig L, Klingel K, Kahlert HA, Priewe J, et al. (2021) Cardiogenic Shock Caused by Cardiac Tamponade Due to Fulminant HHV-6 Myopericarditis In A COVID-19 Patient: A Case Report. Annal Cas Rep Rev: ACRR-200.

Received Date: 07 February 2021; **Accepted Date:** 16 February 2021; **Published Date:** 23 February 2021

Abstract

Background: *COVID-19 is associated with lung involvement up to severe Acute Respiratory Distress Syndrom. It also affects other organs, including the heart. We present a case of a cardiogenic shock caused by a myopericarditis in a COVID-19 positive patient.*

Case Summary: *We present a case of a 38-year old woman without relevant pre-existing illness with a cardiogenic shock caused by a HHV-6 myopericarditis and positive throat test for SARS-CoV2 RNA by absence of pulmonary manifestation. In the CMR a reduced left ventricular ejection fraction was seen, the myocardbiopsy showed the involvement of HHV-6 whereby SARS-CoV 2 RNA quantitative PCR was negative.*

Conclusion: *The detection of SARS-CoV 2 RNA must also be considered in patients without pulmonary symptoms in order to understand the course of the disease. The role of reactivating further viral diseases, in our case HHV-6, seems to be an important point in understanding the disease.*

Keywords: *Cardiogenic shock, COVID-19, Myopericarditis, HHV-6*

Abbreviations

- COVID-19 = Corona Virus Disease 2019
- SARS-CoV-2 = Severe acute respiratory syndrome coronavirus type 2
- HHV-6 = Human Herpesvirus 6
- CT = computed tomography
- CMR = cardiovascular magnetic resonance

Introduction

The World Health Organisation declared Corona Virus Disease 2019 (COVID-19) as a global pandemic on March 11th, 2020 [1]. Primarily, COVID-19 involves the respiratory tract, but several case reports such as meta-analysis also presented a range of cardiac involvement [2] including arrhythmias, acute heart failure, acute coronary syndrome, myocarditis or cardiac arrest [1]. We report a case of a Severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) positive 38-year old woman with a Human Herpesvirus 6 (HHV-6) myopericarditis and a cardiogenic shock caused by cardiac tamponade. We discuss the reactivation of HHV-6 in the myocardium by SARS-CoV2 RNA.

Case Presentation

A 38-year old woman presented to a secondary care hospital with right upper abdominal pain. Dyspnoea, cough, fever, or other symptoms were absent and relevant pre-existing conditions not known. The laboratory tests were inconspicuous. Due to missing laboratory and sonography abdominal pathologies she was initially sent home but presented again the following day because of persistence of complaints. A computed tomography (CT-) scan of the abdomen was performed to rule out cholecystitis and other gastrointestinal causes. During the CT examination hemodynamic instability occurred and the patient was intubated. An emergent CT scan of the chest showed a slight myocardial effusion while pulmonary embolism and aortic dissection as well as characteristics of COVID-19 pneumonia were excluded (Figure 1).

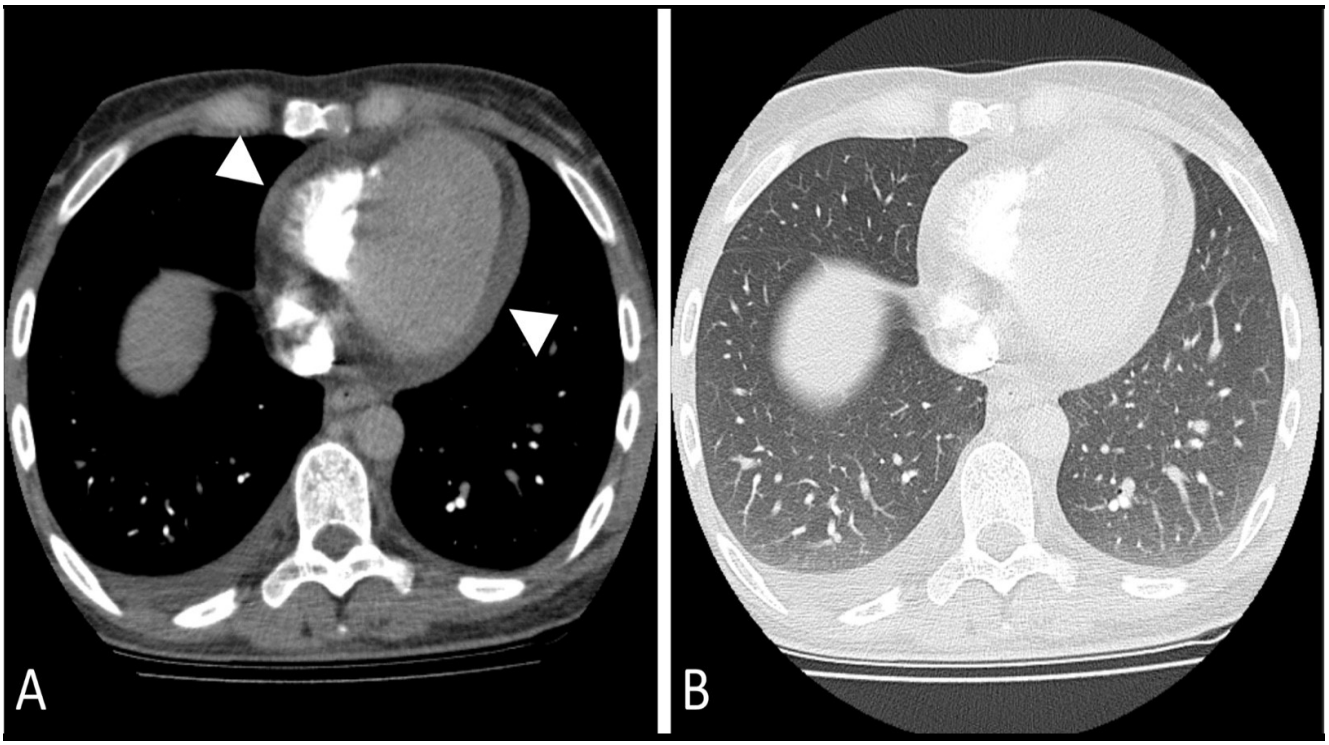

Figure 1: Soft tissue window (A) of pulmonary computed tomography angiography depicts circumferential moderate pericardial (arrowheads) and right-sided pleural effusions. In the lung window settings (B), there were no imaging findings of COVID-19.

Intravenous vasopressor (norepinephrine) therapy was initiated. The ECG showed tachyarrhythmia absoluta and laboratory results showed an increased high sensitivity troponin I (289,2pg/ml, normal <14ng/L), while creatine kinase and myocardial creatine kinase were within normal range. Inflammatory markers and lactate were also increased (leukocytes 14.2 10^3/ul, lactate 6,9mmol/l, normal leukocytes 4,5-11.3x10^3/ul, lactate 0,5-2,2mmol/l). A preliminary assessment by transthoracic echocardiography revealed a reduced ejection fraction with a pericardial effusion without signs of hemodynamic relevance. An electrical cardioversion and intravenous administration of amiodarone were not successful in converting into sinus rhythm. She was emergently transferred to our hospital by helicopter to evaluate her condition for an extracorporeal circulatory life support therapy. During the transport the need for catecholamines increased significantly.

Upon admission to our tertiary care hospital, the ECG showed a sinus tachycardia and the repeated transthoracic echocardiographic examination revealed a pericardial effusion with hemodynamic relevance. Pericardiocentesis was performed immediately with initial drainage of 190cc serous fluid and subsequent drainage of overall 500cc within the next 48 hours. Pericardiocentesis instantly improved hemodynamics and vasopressor therapy could be terminated. The routine testing for SARS-CoV-2 RNA by PCR was positive in the throat swab. For further investigation, cardiovascular magnetic resonance (CMR) was performed and showed a myopericarditis with global hypokinesia and reduced left ventricular ejection fraction (ejection fraction 43%) (Figure 2).

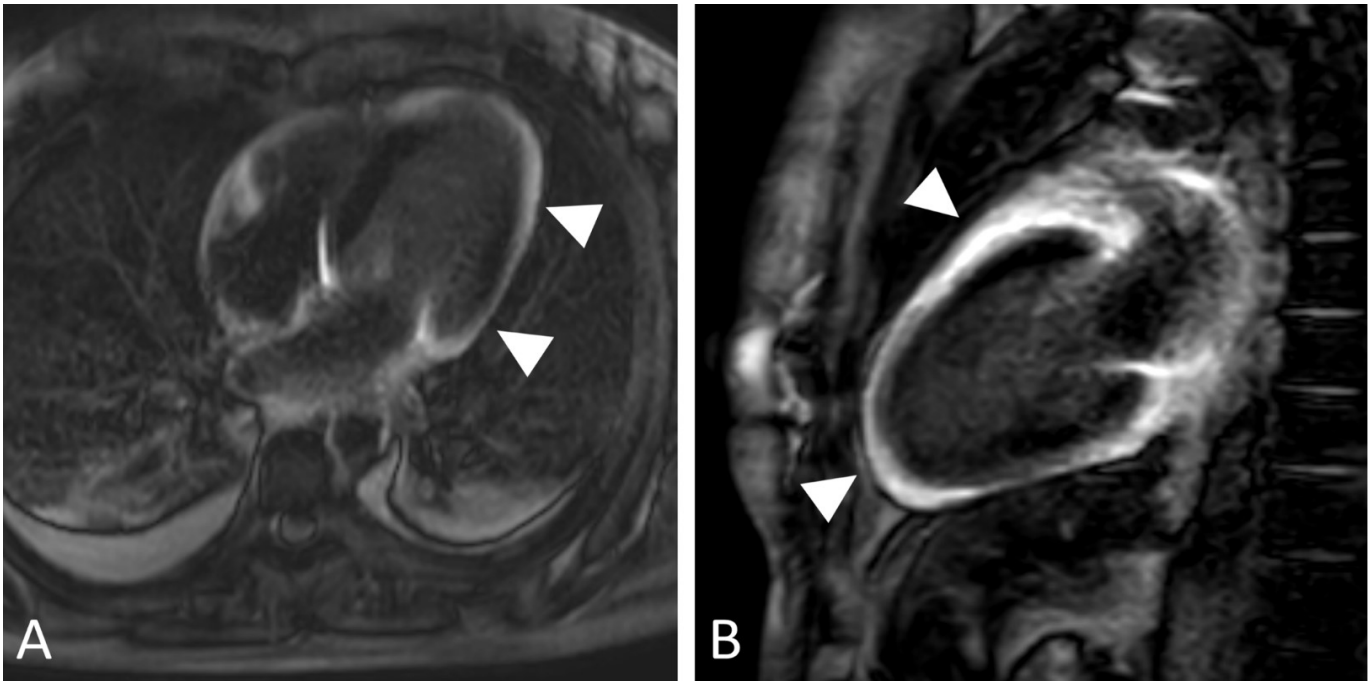

Figure 2: Breath-hold late gadolinium enhancement imaging in 4-chamber (A) and 2-chamber (B) views shows diffuse myopericarditis of the left ventricle (arrowheads). Of note, phase-sensity inversion-recovery technique was employed due to the hampered myocardial nulling in conventional T1 inversion-recovery gradient-echo sequences.

Cytologic analysis of pericardial fluid revealed granulocytes rich irritation effusion without a growth of bacteria. The pericardial fluid was tested negative for SARS-CoV-2 RNA but throat swab tests were repeatedly positive for SARS-CoV-2 RNA without pulmonary symptoms. Furthermore, an endomyocardial biopsy was performed which showed a subacute lymphocytic myocarditis with significantly increased CD3-positive T lymphocytes (up to 62 T cells / mm^2) (Figure 3). The number of CD68-positive macrophages with MHC III expression was moderate. By nested PCR a HHV6B DNA was detected in the heart muscle and the peripheral leukocyte preparation. SARS-CoV-2 quantitative RT PCR was negative in the heart muscle

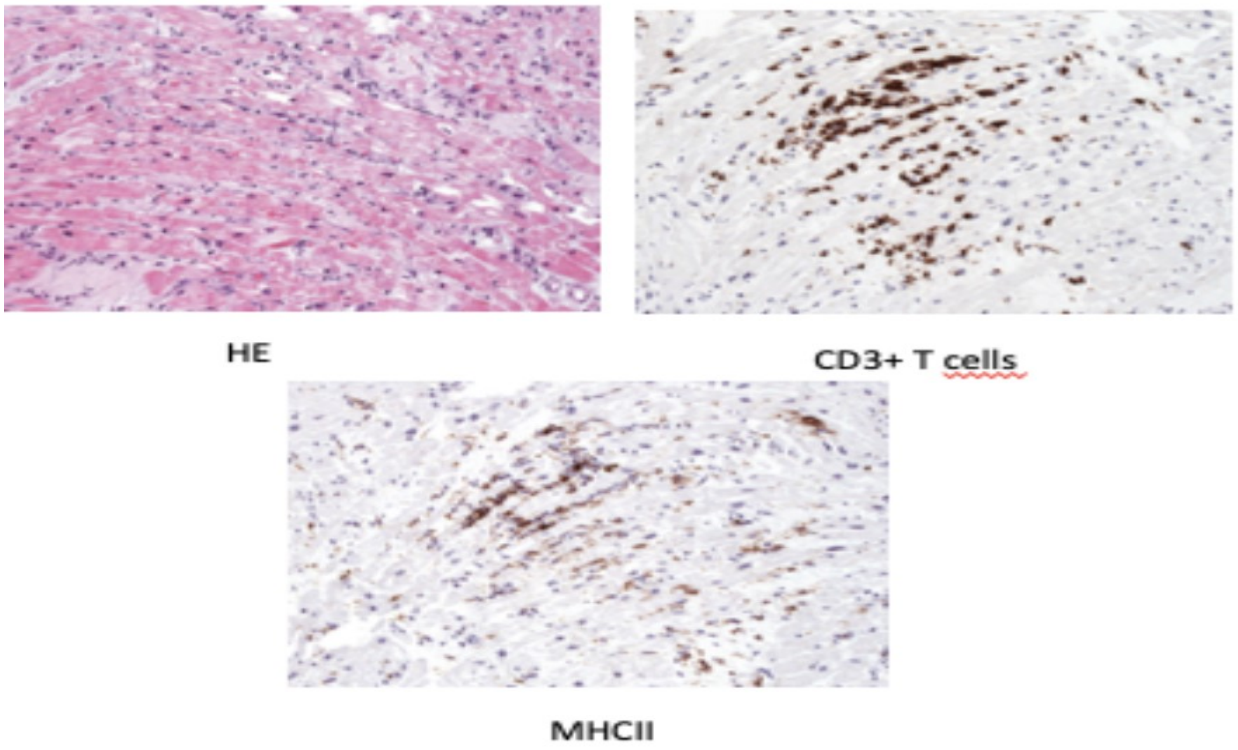

Figure 3: Endomyocardial biopsies showed significantly increased amounts of CD3-positive T lymphocytes and CD68-positive macrophages with MHC II expression.

Discussion

Here, we report a case of a 38-year old woman who developed a life-threatening cardiogenic shock due to a pericardial effusion by lymphocytic myocarditis. She revealed positive throat swab tests for SARS-CoV-2 RNA in absence of pulmonary involvement and in absence of viral RNA in pericardial effusion and heart muscle.

COVID-19 caused by the SARS-COV-2 virus primarily affects the respiratory tract and ranges from no or mild respiratory symptoms to an acute respiratory distress syndrome and multiorgan failure [1]. A systematic review with a meta-analysis of the literature showed that myocardial injury is also found with an incidence of 16% [3]. Another prospective observational cohort study of German patients revealed cardiac involvement in 78% patients as demonstrated by cardiac magnetic resonance [2].

So far, five cases of cardiac tamponade in COVID-19 patients have been reported [4-8]. Four of them had primary respiratory symptoms and one of them developed a pericardial effusion after coronary artery bypass grafting which might not be COVID-19 associated [7]. In our case, both respiratory symptoms and fever were missing and chest-X ray as well as CT scanning showed no characteristics of COVID-19 pneumonia.

The pericardial fluid of our patient was tested negative for SARS-CoV-2. Previously, in only one case report pericardial fluid was tested positive [7]. Furthermore, SARS-COV-2 quantitative RT-PCR in the heart muscle was also negative, but HHV6 DNA was detected in the heart and in the blood suggesting persistence of virus without clinical relevance.

Particularly in acute hemodynamic unstable myocarditis endomyocardial biopsy might help guide advanced medical therapy. Whereas immunosuppression might be applicable when viral presence is excluded or parvovirus B19 is detected al low viral burden, specific antiviral drugs or immunomodulation might be given depending on which virus is detected by PCR [9]. There are currently therapeutic recommendations for pulmonary manifestation of COVID-19 [10], but recommendations for treatment in primary myocardial involvement are lacking. Colleagues from Spain treated a fulminant myocarditis in a COVID-19 patient with immunomodulatory, steroid and immunoglobulin therapy with favorable clinical response [11].

Actually, the pathogenesis of cardiac involvement of SARS-CoV-2 has not yet been fully understood. As both the pericardial effusion and the myocardium did not show presence of SARS-CoV-2 genome, the pericardial effusion might be caused by formation of autoantibodies against myo- and endocardial structures (molecular mimicry) [12]. Moreover, in a case report HHV-6B reactivation in COVID-19 has been documented. It was found that the CD4+ T cells that are present in COVID-19 patients have significantly increased expression of the HHV-6B receptor, CD134 [13]. In our case, the myocarditis could be caused by the reactivation of HHV-6 by SARS-CoV2 RNA.

Conclusion

Although COVID-19 is currently dominating our medical perception, other or coexisting pathologies must not be overlooked particularly in patients with extrapulmonary manifestations. In acute hemodynamic unstable myocarditis, endomyocardial biopsy is important to detect specific pathogens and guide further medical therapy. The role of COVID-19 in cardiac pathologies, even when not directly detectable by PCR, is unclear and needs further study.

Acknowledgement

We know of no conflicts of interest associated with this publication, and there has been no significant financial support for this work that could have influenced its outcome.

Conflict of interest for all contributing authors: None.

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