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Case Report
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Multiorgan Complications in Young Low-Risk Male with Post-COVID-19 Syndrome

Paulina Pikus[1], Marta Jaskólska[1], Radosław Nowak[2], Jadwiga Fijałkowska[3] and Zbigniew Zdrojewski[1]*

[1]Department of Internal Medicine, Connective Tissue Diseases and Geriatrics, Medical University of Gdansk, Poland

[2]Department of Cardiology and Electrotherapy, Medical University of Gdansk, Poland

[3]Second Department of Radiology, Medical University of Gdansk, Poland

Abstract

The COVID-19 pandemic is an ongoing global pandemic with still unknown long-term consequences. Over time, we learn about new possible complications. This case report highlights the possibility of serious complications after mild course COVID-19 infection in young, previously healthy individual.

Case Presentation

A 27-year-old man with no comorbidities was admitted to the clinic due to infection of unknown origin. In the fifth week after the onset of COVID-19 (mild course), the patient reported fever, vomiting, paranasal sinus pain, epigastric pain; for these he was treated with cefuroxime, later changed to ciprofloxacin, with no improvement. The patient subsequently reported visual phenomena which he described as "visions" involving himself (both with his eyes open and closed) accompanied by severe anxiety. The patient was not previously treated psychiatrically. The patient maintained full verbal contact and logical faculties after admission. Upon physical examination, the following were noted: Bilateral tonsillar enlargement, enlargement and tenderness of right cervical lymph nodes, hepatomegaly, and abdominal tenderness.

Laboratory results revealed elevated inflammatory markers (CRP212.29 mg/l, PCT 3.37 ng/L, WBC 9.30 G/L, neut. 8.24 G/L, lymph. 0.51 G/L) and liver enzymes (ALT 82 U/L, GGT 225 U/L, BIL 33.52 umol/L), an increase in hsTnI 0.0506->0.0648 ng/ml, BNP 61 pg/ml. In ECG sinus tachycardia was observed. Moreover, the patient was tested for SARS-CoV-2 infection - PCR antigen test negative, positive IgG antibodies and negative IgM antibodies. A thoracic and abdominal CT revealed hepatosplenomegaly, and hepatic hilar lymphadenopathy. A hepatotropic viral infection was excluded. No abnormalities in the lungs were detected (CORADS 1).

During hospitalization, empiric antibiotic therapy was continued (ceftriaxone, clarithromycin).

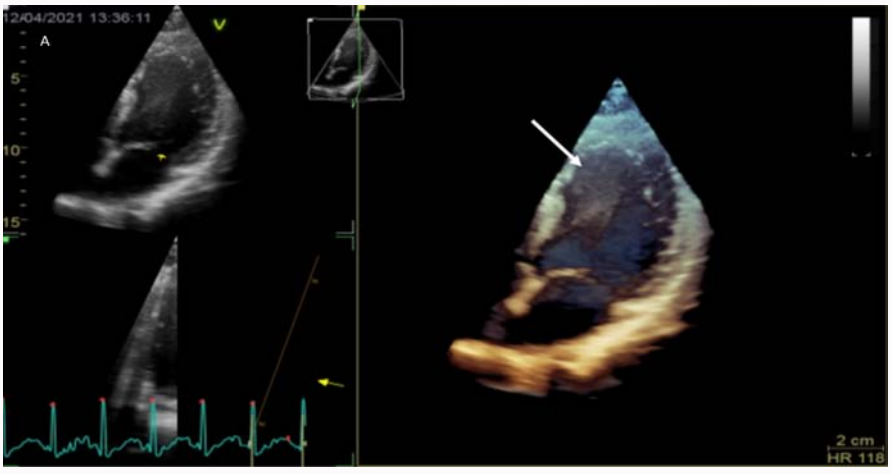

Figure 1: 3D transthoracic echocardiogram of left ventricular apical sludge.

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Correspondence:
Marta Jaskólska, Department of Internal Medicine, Connective Tissue and Geriatrics, Medical University of Gdansk, ul. Dębinki 7, 80-952 Gdańsk, Poland, Tel: +48-583492512; E-mail: marta.jaskolska@gumed.edu.pl

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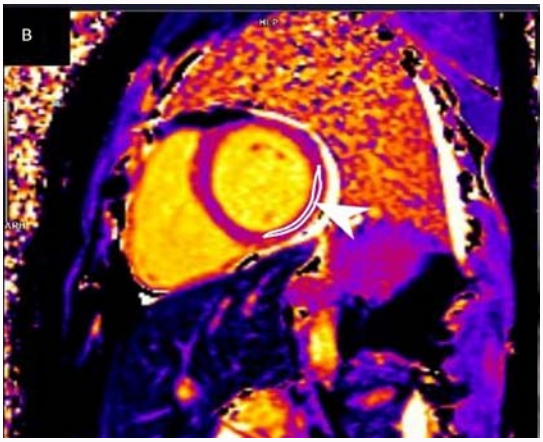

Figure 2: T1 map with color overlay mid short-axis showing significantly raised native T1 (elevated T1, 1112 milliseconds) indicated by white arrowheads.

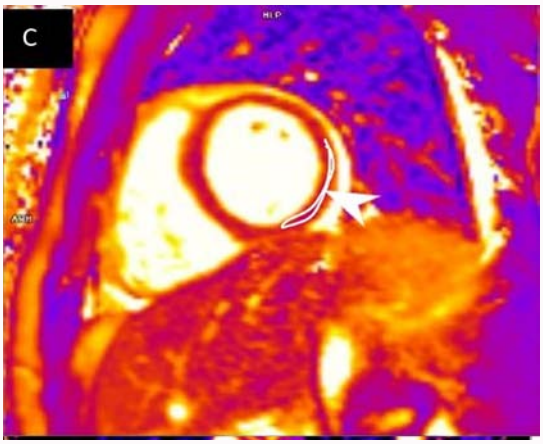

Figure 3: T2 map with color overlay mid short-axis showing myocardial edema (elevated T2, 54 milliseconds) indicated by white arrowheads.

The patient suffered several bouts of fever. Blood cultures, throat swab, respiratory PCR panel, and serodiagnostic tests for infectious causes were all negative.

A panoramic radiograph was performed; tooth number 18 was extracted. A paranasal sinus X-ray and otolaryngological examination did not reveal abnormalities that could correspond with the increased inflammatory markers.

Transthoracic Echocardiographic examination (TTE) revealed mildly reduced Left Ventricular Ejection Fraction (LVEF) and unusual LV apical sludge as a result of hypercoagulable state (Figure 1). Anticoagulants were prescribed. Cardiac MRI confirmed a localized active inflammatory process (Figure 1). It was decided that the patient would continue treatment with anticoagulant, ACE-I, B-blocker and ivabradine.

The consulting psychiatrist put forward a diagnosis of post-COVID neurasthenia and prescribed pernazinum and diazepam. Neuroinfection was excluded based on MRI of the brain and lumbar puncture (cerebrospinal fluid analysis within norm, negative PCR neuro panel).

During the hospitalization the clinical symptoms (fever, psychiatric symptoms) resolved and the inflammatory parameters

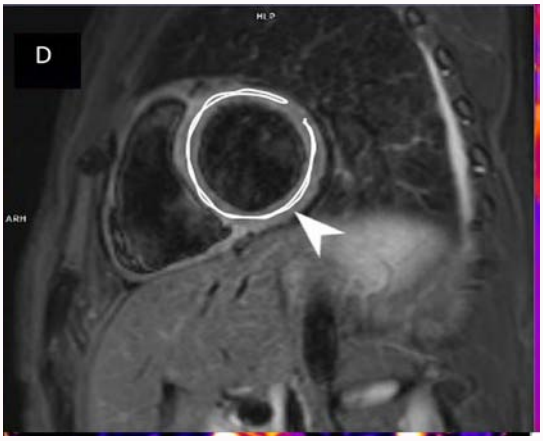

Figure 4: T2-weighted fat-saturated double inversion recovery fast spin echo short-axis image showing myocardial edema.

returned to normal. Control TTE showed complete sludge resolution. Taking into account the overall clinical picture, it was concluded that the observed abnormalities and symptoms were a result of the post-COVID syndrome.

Discussion

A substantial psychiatric morbidity has been demonstrated in convalescents, especially those with severe COVID-19 [1]. The pathomechanism may be related to secondary abnormal host immune response and cytokine network deregulation [2]. In this case, hemostasis disorders could play an additional role. The link between COVID-19 and coagulopathy was described in several studies [3]. Regarding cardiovascular complications, Bailey et al. [4] concluded that SARS-CoV-2 can infect human cardiomyocytes causing sarcomere breakdown, necrosis, and reduced contractility. Myocardial injury may also affect convalescents, regardless of pre-existing conditions, severity, and the overall course of the acute illness [5].

Patients reporting new symptoms after recovering from COVID require careful clinical evaluation because severe post-COVID-19 syndromes may occur in young patients with mild disease course. Due to possible involvement of many organs extensive and interdisciplinary diagnostics is necessary.

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