

MEDICAL HISTORY AND LABORATORY TESTS ARE CRUCIAL TO ELUCIDATE THE TRIGGERS OF TAKOTSUBO IN COVID-19

Josef Finsterer¹, Claudia Stöllberger²

¹ Neurology & Neurophysiology Center, Vienna, Austria

² 2nd Medical Department, Klinik Landstrasse, Vienna, Austria

Corresponding author: Josef Finsterer e-mail: ffigs1@yahoo.de

Received: 22/11/2023 Accepted: 27/11/2023 Published: 22/12/2023

Conflicts of Interests: The Authors declare that there are no competing interests.
This article is licensed under a [Commons Attribution Non-Commercial 4.0 License](#)

How to cite this article: Finsterer J, Stöllberger C. Medical history and laboratory tests are crucial to elucidate the triggers of Takotsubo in COVID-19. *EJCRIM* 2023;10:doi:10.12890/2023_004223.

KEYWORDS

Type A aortic dissection, Bentall procedure, atypical symptom

LETTER TO THE EDITOR

We read with interest Mohammed et al.'s article^[1] about a 60-year-old female who developed classic Takotsubo syndrome (TTS), which was due to emotional stress from her work as a health worker in a senior care home, where several residents were infected with SARS-CoV-2 and some died from the infection. The patient's professional responsibilities multiplied due to the quarantine of many of her colleagues^[1]. It was concluded that TTS may result from emotional stress and that recognition of TTS requires knowledge of a patient's living environment, mental health and daily stresses^[1]. The study is appealing but raises the following comments and concerns.

The main limitation of the study is that it remains unclear whether the patient was ever infected with SARS-CoV-2. The uncertainty is further reinforced by the question in the title^[1]. Although the index patient tested negative for SARS-CoV-2 upon admission, it cannot be ruled out that the patient was previously infected with SARS-CoV-2 without developing classic pulmonary manifestations. Therefore, we should know whether the index patient tested positive for the presence of antibodies against SARS-CoV-2. A subclinical infection is conceivable because she initially worked unprotected with COVID-19 patients, and together with

colleagues who had to be quarantined due to an infection. Since COVID-19 is frequently complicated by a cytokine storm manifesting as multisystem inflammatory syndrome (MIS)^[2], we should know the serum levels of C-reactive protein, D-dimer, ferritin, interleukin (IL)-6, and the leukocyte and thrombocyte counts of the index patient. Because cardiac disease may be the initial manifestation of SARS-CoV-2 infection^[3] and because cardiac complications may occur in elderly patients with high inflammatory markers^[4], it is important that TTS be considered a manifestation of COVID-19 in the index patient. A negative PCR test from a nasopharyngeal swab does not necessarily mean that the patient was not infected^[5]. The sensitivity of PCR tests for SARS-CoV-2 varies depending on the product and is usually not 100%. The risk of a negative PCR test is particularly high in patients with high inflammatory markers^[6]. Because the patient suffered from exertional dyspnoea a week before admission, it is critical that SARS-CoV-2 associated pneumonia was properly ruled out. Since the patient had suffered a disorientation episode the day before admission^[1], it is important that she was examined by a neurologist and underwent cerebral MRI and electroencephalography. Assuming that the TTS had already begun a week before presentation^[1], it is conceivable that

the patient had suffered a transitory ischaemic attack (TIA) or a minor stroke due to low output failure and intracardiac thrombus formation. It is known that TTS is associated with an increased risk of ischaemic stroke^[7]. It is also conceivable that the patient had suffered non-convulsive seizures triggered by low serum sodium levels secondary to sweating, the day before presentation.

Overall, this interesting case report has several limitations that should be addressed before attributing the development of TTS to emotional stress. Since MIS following infection with SARS-CoV-2 has not been sufficiently ruled out, TTS triggered by the immunological reaction to the virus cannot be definitively ruled out.

REFERENCES

1. Mohammed M, Zakhour S, Devgun J, Lee J, Keimig T, Wang DD. Takotsubo cardiomyopathy in a healthcare worker during the COVID-19 pandemic: caused by the virus or the demands of the many being placed on the few? *Eur J Case Rep Intern Med* 2020;7:002088.
2. Ahsan T, Rani B. A case of multisystem inflammatory syndrome post-COVID-19 infection in an adult. *Cureus* 2020;12:e11961.
3. Cairns L, Abed El Khaleq Y, Storrar W, Scheuermann-Freestone M. COVID-19 myopericarditis with cardiac tamponade in the absence of respiratory symptoms: a case report. *J Med Case Rep* 2021;15:31.
4. da Silva JF, Hernandez-Romieu AC, Browning SD, Bruce BB, Natarajan P, Morris SB, et al. COVID-19 clinical phenotypes: presentation and temporal progression of disease in a cohort of hospitalized adults in Georgia, United States. *Open Forum Infect Dis* 2020;8:ofaa596.
5. Yan CH, Prajapati DP, Ritter ML, DeConde AS. Persistent smell loss following undetectable SARS-CoV-2. *Otolaryngol Head Neck Surg* 2020;163:923–925.
6. Lascarrou JB, Colin G, Le Thuaut A, Serck N, Ohana M, Sauneuf B, et al. Predictors of negative first SARS-CoV-2 RT-PCR despite final diagnosis of COVID-19 and association with outcome. *Sci Rep* 2021;11:2388.
7. Morris NA, Chen ML, Adejumo OL, Murthy SB, Kamel H, Merkler AE. Stroke risk following Takotsubo cardiomyopathy. *Neurohospitalist* 2020;10:277–280.