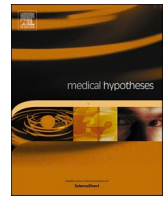




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Letter to Editors

SARS-CoV-2 associated polyradiculitis and myocarditis may favour Takotsubo syndrome

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With interest we read the article by Szarpak et al. about considerations on the relation between lung disease due to an infection with SARS-CoV-2 (COVID-19) and stress cardiomyopathy, also known as broken heart syndrome, ampulla cardiomyopathy, apical ballooning syndrome, Takotsubo cardiomyopathy, or Takotsubo syndrome (TTS) [1]. At least four types of TTS are differentiated, the apical and most frequent type, the mid-ventricular type, the basal type, and the global type [2]. TTS is due to physical stress or emotional stress in one third of the cases each, but the cause remains elusive in the remaining third of patients. Szarpak et al. hypothesise that the prevalence of TTS has increased during the pandemic due to an evident increase in individual or collective emotional stress within societies [1]. The valuable considerations can be supplemented by further thoughts on the pathophysiology of TTS in COVID-19 patients.

TTS has been hypothesised to result not only from elevated levels of catecholamines and its metabolites, microvascular dysfunction, estrogen deficiency, spasms of the epicardial coronary arteries, or aborted myocardial infarction, but also due to myocardial inflammation or autonomic dysfunction [3]. On the other hand, an infection with SARS-CoV-2 may not only manifest in the lungs but also extra-pulmonary in the central nervous system (CNS), peripheral nervous system (PNS), eyes, guts, liver pancreas, kidneys, heart, muscles, and skin [4]. Since one of the most frequent cardiac manifestations of COVID-19 is myocarditis [5] it is conceivable that the increased level of pandemic stress may easily trigger TTS in patients infected with SARS-CoV-2 and myocardial inflammation. Whether TSS is more prevalent in patients with sepsis than those without, remains unknown so far.

A PNS manifestation of COVID-19 is polyradiculitis also termed Guillain-Barre syndrome (GBS) [6]. Since GBS may affect also the autonomic fibers, it is conceivable that denervation of the myocardial autonomic innervation may favour the development of TTS in COVID-19 patients under emotional or physical stress due to respiratory failure requiring artificial ventilation, pain, or anxiety.

Szarpak et al. support their hypothesis about the putatively increased frequency of TTS since onset of the pandemic by listing a number of studies and case reports. The list of reports is incomplete [7] and the question if the frequency of TTS has truly increased compared to periods

prior to the pandemic remains unanswered. Currently, there are only few studies available which compare the prevalence of TTS in COVID-19 patients with TTS from a previous periods. In a single centre study from France the incidence of TTS did not increase when compared with the years 2016 to 2019 [8]. Single cases do not support the hypothesis and comparative, large cohort studies are required to solve the issue.

Overall, the interesting hypothesis raised by Szarpak et al. has not been substantiated yet. Myocarditis caused by SARS-CoV-2 and GBS caused by the immune response to the virus should be considered as pathophysiological conditions favouring the development of TSS in patients infected with SARS-CoV-2.

Declarations

Statement of ethics: was in accordance if ethical guidelines.

Conflicts of interest: none.

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Author contribution: JF: design, literature search, discussion, first draft, critical comments, final approval, FS: literature search, discussion, critical comments, final approval.

Informed consent: was obtained.

The study was approved by the institutional review board.

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