STRESS CARDIOMYOPATHY IN COVID-19 DISEASE

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Abstract: *Takotsubo syndrome (TTS), also known as stress cardiomyopathy, can occur in COVID-19 pneumonia patients at various stages of the illness and with varying degrees of left ventricular failure. We outline three TTS cases involving COVID-19-positive individuals with various clinical outcomes. One of them passed away, and in the other two, coronary angiography confirmed the diagnosis but was delayed due to the potential for virus spread until after the pneumonia had cleared up.Learning points It is underlined that COVID-19 and cardiac involvement are related. Because Takotsubo syndrome is thought to be brought on by extreme stress, its prevalence has increased throughout this epidemic.*

Key words: *Takotsubo syndrome, cardiomyopathy, COVID-19*

INTRODUCTION

Takotsubo syndrome (TTS), a stress cardiomyopathy that causes quick, severe, and typically reversible cardiac failure as a result of significant mental or physical stress, affects 1.8–2.2% of all acute coronary syndromes [1]. There are theories that TTS and COVID-19 may be related.

CASE DESCRIPTION

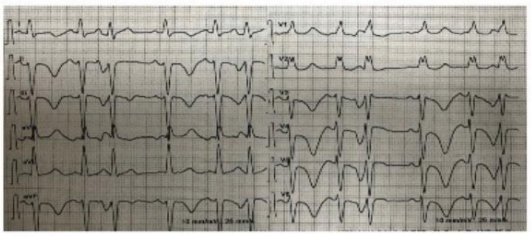
In this article, we discuss three instances of stress cardiomyopathy in patients who were recently admitted to the Covid hospital in Dolo, Italy, close to Venice, for a severe case of acute respiratory syndrome brought on by COVID-19 infection. According to the Clinical Frailty Scale, they were all in good clinical condition before to hospitalization and had histories of arterial hypertension or diabetes that had been adequately managed. They all experienced common signs of infection, such as fever, cough with dyspnea, and unusual chest discomfort in the 10 days before to going to the emergency department (ED), but none of them had a history of cardiovascular disease.

CRP, C-reactive protein; HF, high flow; Hs-cTn, high sensitivity troponin T; iv, intravenous; sc, subcutaneous. In the emergency department, COVID-19 was detected in all three nasopharyngeal samples. After patients were hospitalized, their clinical state was evaluated for changes, and blood samples were examined for high-sensitivity troponin T (hs-cTn), NT-proBNP, and D-dimer. Antiviral medications and hydroxychloroquine were administered to all three patients right away, and a daily EKG was done to assess the length of the QT interval. All three patients' pulmonary CT scans revealed bilateral lung consolidation and ground-glass opacities. None of the patients reported symptoms, EKG findings, high hs-cTn values, or echocardiographic findings that suggested ischaemia, but

all had high D-dimer values or clinical characteristics that indicated thromboembolic acute episodes.In addition to other treatments required by hospital protocol, subcutaneous fondaparinux 2.5 mg was given to COVID-19 patients because there was evidence of hypercoagulation in these individuals.

Based on EKG abnormalities with diffuse negative T waves on precordial leads and QT interval prolongation (Fig. 1) and a typical echocardiographic pattern with dyskinesia of the left ventricle apex (apical ballooning) and basal wall hypercontractility with systolic dysfunction, a stress cardiomyopathy was suspected.

Figure 1



EKG of patient 1

The first patient received high-flow oxygen therapy with a nasal cannula and was an 84-year-old man with a normal PaO2/FiO2 ratio. He experienced sudden exacerbation of his dyspnea and chest pain while in the hospital, and his blood pressure (BP) was measured at 220/100 mmHg. Following early administration of nitroglycerin and metoprolol intravenously, the patient's symptoms gradually improved and their blood pressure returned to normal. Although hs-cTn was elevated (70 ng/ml), it did not exhibit the usual ischemic rise. A typical TTS was seen on the EKG and ultrasound, with a global left ventricular ejection fraction (EF) of 53% preserved.Coronary angiography was delayed for a few weeks until pneumonia had fully recovered due to extensive COVID-19 lung involvement, in order to prevent potential cardiovascular consequences and virus dissemination. Prior to coronary angiography, which revealed no substantial coronary stenosis, aspirin was started.

A second patient, an 85-year-old woman with fever and a cough, was seen in the Covid department. Two days later, she developed septic shock from a Pseudomonas aeruginosa infection and was sent to the intensive care unit. She needed mechanical ventilation due to progressive respiratory failure (PaO2/FiO2 100), but the levels of blood oxygen saturation did not rise. As a result, we conducted an echocardiography and an EKG, both of which were extremely suspicious for TTS. The echocardiogram revealed a substantial (30%) decline in LVEF. The patient died within a few hours despite ventilation while lying on his or her back, the use of a plasma expander, and inotropic support. A normal coronary anatomy was established by the autopsy.

The third patient, an 81-year-old male maintained with high-flow nasal cannula oxygen, had a normal PaO2/FiO2 ratio. High NT-proBNP levels and aberrant hs-cTn values were found in blood samples, but there was no rise in these markers of ischaemia. A

substantially compromised LVEF (42%), along with usual TTS changes, were seen on the EKG and echocardiogram. After the pneumonia had cleared up, a coronary angiography was done, but it revealed no substantial coronary stenosis.

DISCUSSION

Stress cardiomyopathy has not been widely reported in the COVID-19 group. Direct myocardial injury, however, has frequently been reported and linked to coronary artery disease, hypoxia, or myocarditis. Additionally mentioned as side effects of COVID-19 infection include coagulopathy and vascular endothelial dysfunction [4].

Extreme sympathetic activation, aberrant catecholamine release, and consequent epicardial coronary spasm are the typical causes of stress cardiomyopathy [5-7]. In COVID-19 patients, a number of distinct pathways coexist that could be a factor in the development of left ventricular dysfunction. This pandemic illness may cause stress, which could lead to an increase in stress cardiomyopathy cases. We also want to stress that the potential of virus dissemination through rapid coronary angiography in acute COVID-19 pneumonia could affect both the patient and the cardiologist.We should maintain a suspicion of stress cardiomyopathy and defer invasive testing until after full recovery from COVID-19 pneumonia since it's possible that patients with the virus may have acute coronary syndrome.

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