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Editorial Commentary

Acute, Transient Cardiomyopathy in Patients With COVID-19: Can We Clearly and Quickly Diagnose Its Cause and Nature?

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In this issue of the *Journal*, Yalamanchi et al[1] describe the case of a young male patient with COVID-19 who appeared to recover quickly but soon developed acute congestive heart failure with a left ventricular ejection fraction of 20% and a global pattern of critical hypokinesia. This case is clearly important, but the presentation and explanation of the possible pathophysiologic mechanisms are confusing.

Apparently, the patient recovered relatively quickly and had a near-normal left ventricular ejection fraction in 1 month. The most likely diagnosis in such a case—transient takotsubo cardiomyopathy (TTC)—should have been quickly ruled out. The reported testing seemed to have ruled out myocardial scarring (coronary embolism and myocarditis) but not TTC. The early disappearance of severe cardiomyopathy is pathognomonic of TTC by itself.

At this time, in similar cases (which are generally rare but encountered fairly frequently at specialized centers), acetylcholine testing is needed urgently to identify the specific cause of patient's endothelial dysfunction.[2] Preliminary results suggest that acetylcholine testing for this indication is feasible, safe, and specific for endothelial dysfunction (when the test result is positive). Thus, acetylcholine testing seems to be a good option in any case of either acute ischemic syndrome with cardiomyopathy or myocardial infarction with nonobstructive coronary arteries or of acute myocardial infarction with a "normal coronary tree."

One of the most convincing current theories regarding TCC claims that its root cause is endothelial dysfunction (in cases of COVID-19, endothelial dysfunction resulting from endotheliitis caused either by direct viral infection or inflammatory disease).[3,4] The biological state characterized by endothelial dysfunction is not yet clearly understood, but it seems a logical reason to induce coronary spasm with acetylcholine, especially in a patient with stress, respiratory distress, anxiety, and fever caused by COVID-19. In similar cases, endothelial dysfunction manifests as an acute event (typically with chest pain and dyspnea) and leads quickly to left ventricular dysfunction. With advanced testing, the authors ruled out both myocarditis and acute myocardial infarction.[1] An early acetylcholine test for endothelial dysfunction would have been a simple and safe alternative, but it requires specialized techniques that may not be available in all catheterization laboratories. Such testing facilitates reliable documentation of a reproducible mechanism (coronary artery spasm) and response to nitroglycerine.

In addition, this unfortunate patient was in a severely hypercoagulable state, with multiple systemic embolisms. Most likely, the emboli originated in the left ventricle, as indicated by dimerized plasmin fragment D elevation and pointing to TTC. Recent evidence suggests that TTC occurrence has been 4 to 5 times more frequent during than before the COVID-19 pandemic.[4]

Acetylcholine testing is indicated for establishing a diagnosis and selecting treatments for individual patients. It also creates opportunities to collect useful data from large TTC populations.

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