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E-Challenges & Clinical Decisions

COVID 19: A Stressor for Both the Patient and the Anesthesiologist

Terrence F. Feldheim, MD*, Margot Denham, MD†, Wanda M. Popescu, MD†,‡,

*Yale New Haven Hospital, New Haven, CT
†Yale School of Medicine, New Haven, CT
‡Veteran’s Administration Connecticut Healthcare System, West Haven, CT

The coronavirus disease 2019 pandemic has created not only widespread morbidity and mortality, but a myriad of social, financial, and psychological stressors. In this setting, the medical community has seen a substantial increase in the incidences of cardiac morbidity and mortality, and, therefore, anesthesiologists should expect a higher incidence in the perioperative period. In this E-Challenge, the authors present a patient in whom an acute cardiac decompensation occurred secondary to an unanticipated difficult intubation, with an unexpected echocardiographic finding.

Key Words: acute coronary syndrome; coronavirus disease 2019; COVID-19; heart failure; left ventricular ejection fraction

Background

THE EFFECT that coronavirus disease 2019 (COVID-19) has had on the medical community is incalculable. The pandemic has presented new challenges daily, not only medical, but economic, social, and psychological. As the understanding of this novel disease evolves, new insights into medical management and both short- and long-term repercussions continue to be discovered. The medical sequelae are not limited to those who have contracted the COVID-19 virus; those living in the new world of a global pandemic have been affected in surprising ways. In this E-Challenge, the authors present a case of an unanticipated medical consequence in the setting of the COVID-19 pandemic.

Clinical Case

A 74-year-old male, 104 kg and 172 cm, presented in June 2020 for a scheduled right carotid endarterectomy for high-grade (90%) internal carotid artery stenosis that was asymptomatic. Surgery had been postponed since March 2020 as a result of the COVID-19 pandemic. The medical history was significant for hypertension, hyperlipidemia, type II diabetes mellitus, and obesity. The National Surgical Quality Improvement Program surgical risk calculator identified the patient at a 1.2% risk for cardiovascular complications. A preoperative transthoracic echocardiogram, performed three months prior, revealed a completely normal examination (Video 1). Electrocardiogram (ECG) showed normal sinus rhythm (NSR) with left-axis deviation and poor R-wave progression. The patient demonstrated an exercise tolerance greater than four metabolic equivalents. Preoperative COVID-19 testing was negative.

On the day of surgery, the preoperative airway examination was notable for Mallampati class III; thyromental distance of 3 cm; large, thick neck with redundant tissue; and facial hair. The rest of the physical examination and review of symptoms were unremarkable. The patient denied any new-onset chest pain, shortness of breath, or other concerning symptoms.

The patient was brought to the operating room and placed in a ramped position on the procedure table. Standard American Society of Anesthesiologists monitors were applied. After
thorough preoxygenation, induction of anesthesia was performed with 100 μg of fentanyl, 100 mg of lidocaine, 150 mg of propofol, and 80 mg of rocuronium. The patient required two-handed mask ventilation with an oral airway. Intubation initially was attempted using a video laryngoscope with a size 4 blade. Upon entry into the oropharynx, no identifiable anatomy was noted, but rather extensive redundant tissue. In order to establish a controlled airway, multiple intubation attempts were required, with the utilization of various airway management devices. During this time, the patient became significantly tachycardic and hypertensive, requiring multiple propofol, fentanyl, and esmolol boluses. Throughout the intubation attempts, several episodes of non-sustained ventricular tachycardia were noted, which subsequently progressed to a supraventricular tachycardia, with heart rates between 120 and 150 beats per minute. An intraoperative 12-lead ECG identified new-onset atrial fibrillation, with no ST-segment changes. Incremental doses of metoprolol were administered in an attempt to reduce the heart rate and convert the patient to NSR. To evaluate the patient’s intraoperative cardiovascular status, a transesophageal echocardiographic examination (TEE) was performed (Videos 2-4).

**E-Challenge**

Based on interpretation of the TEE videos, what is the most likely etiology for this patient’s cardiovascular decompensation? What about the time period of the COVID-19 pandemic makes this finding more likely?

**Clinical Course**

A rescue, time-constrained, intraoperative TEE examination was performed, and, therefore, the ECG is absent in Videos 3 and 4. The echocardiographic examination revealed markedly reduced left ventricular (LV) systolic function, with a visually estimated ejection fraction (EF) of 15%-to-20%. Despite the LV foreshortening seen in Video 3, the apex is notably akinetic, the basal segments appear more contractile and the left atrium appears dilated. Fig 1 illustrates the left ventricle in end-systole in the midesophageal two-chamber view. The arrows indicate the classic sign of “apical ballooning” observed in Takotsubo cardiomyopathy (TC) patients. Given the acute changes in LV function and the new onset of atrial fibrillation with a high ventricular rate, the decision was made among the anesthesiology team, the surgical team, and a cardiology consultant to cancel the planned procedure. The patient remained intubated and was transferred to the intensive care unit (ICU) on a propofol infusion for sedation.

The ICU team, in concert with cardiology, believed that the patient’s presentation merited non-urgent evaluation for a coronary syndrome and scheduled a cardiac catheterization the next morning. While awaiting this, his ICU course was remarkable for a spontaneous conversion to NSR. A transthoracic echocardiogram at that time revealed, despite conversion to NSR, only a mild improvement in his EF to 30%, with persistent apical akinesia and hyperdynamic basal segments (Video 5). Troponins T were trended and they peaked at 0.530 ng/mL on postoperative day zero and then returned to normal.

On postoperative day one, the left-sided heart catheterization identified only nonobstructive coronary artery disease. Given the patient’s severely reduced EF, with regional wall motion abnormalities (RWMAs) consistent with apical ballooning (see Fig 1), mildly elevated troponin levels, and nonobstructive coronary disease, the patient was indeed diagnosed with stress-induced (Takotsubo) cardiomyopathy.

The patient was started on a heparin infusion, aspirin, and a combination of sacubitril (neprilysin inhibitor) and valsartan (angiotensin II receptor blocker). He was discharged home within 48 hours of admission. A repeat echocardiogram four weeks after discharge was notable for a full recovery of the EF to 60% and normalization of all RWMAs (Video 6). Six weeks later, the patient underwent an uneventful transcarotid artery revascularization under general anesthesia. For that procedure the airway was secured via an awake bronchoscopic intubation.

**Discussion**

TC is a rare syndrome of acute, reversible heart failure in patients presenting with symptoms suggestive of acute coronary syndrome (ACS). It typically is described in postmenopausal women and triggered by major stressor events. TC remains a diagnosis of exclusion, and each patient warrants a timely and thorough workup for coronary ischemia. Diagnostic criteria for TC were defined in 2014 by the International Expert Consensus Document on Takotsubo Syndrome.¹ Echocardiographic examination represents a cornerstone of the diagnosis. The classic feature of “apical ballooning” consists of transient hypokinesis, akinesis, or dyskinesis of the left ventricle, usually involving the apical and mid-segments.² Rare cases of basal ballooning have been...
described and termed “reverse Takotsubo.” In addition, in very rare instances, a midventricular form of TC and a focal form, mostly involving the anterolateral wall, have been described. Right ventricular involvment is characterized by dilation with hypokinesia of the free wall and the apex. Generally, RWMAs cross multiple coronary perfusion territories. In patients with a prominent septal bulge, as a result of the hypercontractile basal segments, LV outflow tract (LVOT) obstruction and systolic anterior motion of the mitral valve leaflets with subsequent mitral regurgitation are observed. However, mitral regurgitation also can be present as a result of the leaflet tethering related to papillary muscle dysfunction. Severe obstructive coronary pathology or acute plaque rupture are not identified during cardiac catheterization. However, patients with TC often have a concomitant diagnosis of coronary artery disease. Speckle-tracking echocardiography may be helpful in distinguishing the global aspect of TC versus a more regional distribution found with coronary artery occlusion. Patients with TC evaluated with speckle-tracking echocardiography present with a typical “circular” systolic dysfunction. A paradoxical positive longitudinal systolic strain of biventricular mid-apical segments also is identified.

ECG changes, such as ST-segment elevation or depression, nonspecific T-wave inversions, QT prolongation, or low QRS voltage, are common findings in TC. A moderate elevation in serum cardiac troponin and creatinine kinase and a more profound increase in plasma beta-natriuretic peptide levels are seen. Nevertheless, the increase in serum myocardial necrosis biomarkers is disproportionally low compared with the severity of the RWMA, suggesting a reversible nature of injury. Cardiac magnetic resonance imaging identifies the presence of cardiac edema and the absence of late gadolinium enhancement. The presence of infectious myocarditis excludes the diagnosis of TC. Interestingly, TC recently was described in COVID-19–positive patients. The pathophysiology of TC in this context appears to be related to the direct “toxic” effect of cytokines on the cardiomyocyte. The incidence of TC among patients with symptoms of ACS generally has been 1%-to-2%. However, a recent large retrospective cohort study showed an increased incidence of TC during the COVID-19 pandemic. In that study, patients presenting for ACS during March and April 2020 had a 7.8% incidence of TC as diagnosed by echocardiographic features in the context of nonobstructive coronary disease observed during cardiac catheterization. It is important to note that all patients diagnosed with TC in that study tested negative for COVID-19. Therefore, the authors suggested that the social, financial, and psychological stressors posed by the COVID-19 pandemic itself have created the perfect milieu for the development of TC.

Analysis of the etiology of TC has shown that physical stress (35%-55%) is cited more often than emotional stress (20%-39%) as an inducing event. In the setting of this global pandemic, the authors of the present report suspect that there may be a two-hit phenomenon contributing to the increased incidence of TC. The social, psychological, and economic strife facing many patients may lead to an increased baseline catecholamine state and ultimately lower the threshold for additional insults (physical or psychological in nature) to incite the debut of TC. In the case presented herein, for a patient whose important surgery was delayed for months because of the COVID-19 pandemic, the unanticipated difficult intubation served as a “second hit” to trigger intraoperative stress-induced cardiomyopathy.

While anesthesiologists are on alert for complications of COVID-19 that may affect their patients, it is important to understand that factors beyond the simple positive or negative COVID test can lead to poor perioperative outcomes. The patient described herein, with a normal preoperative EF, underwent the stress of surgical postponement and social isolation imposed by the COVID-19 pandemic, followed by several intubation attempts at induction of anesthesia, and developed an acute significant decrease in global ventricular function with new-onset atrial fibrillation with a rapid ventricular response. These findings merited cancellation of the planned procedure and evaluation for ACS.

The management of stress-induced cardiomyopathy consists of primarily providing supportive care until recovery of ventricular function. In patients with none or mild signs of heart failure, initiation of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers and beta-blockers is recommended. However, in patients exhibiting signs of heart failure, diuretics and nitroglycerine could be added to the therapeutic regimen provided that LVOT obstruction is not present. Patients presenting with hypotension or cardiogenic shock initially should be evaluated for the presence of LVOT obstruction. In patients with LVOT obstruction, gentle hydration with intravenous fluid and cautious use of short-acting beta-blockers can be considered. Patients in cardiogenic shock may require temporary circulatory support. An important consideration for perioperative physicians is that, in all patients with TC, the use of inotropic medications should be avoided because they may worsen the cardiomyopathy as a result of the high catecholamine state seen in the disease process. Anticoagulant therapy can be considered in patients with decreased EF or arrhythmias.

The overall prognosis of TC is good, with 96% of patients exhibiting a full cardiac recovery. However, TC is not a completely benign disease process, incurring a mortality of 1%-to-2%. Heart failure and pulmonary edema are the most common complications. Recurrence involves 5%-to-10% of patients and typically is seen within the first month of the initial event.

Conclusion

Given the new data pertaining to the increased risk of developing stress-induced cardiomyopathy during this unprecedented period, the authors of the present report believe that it is of critical importance to alert perioperative clinicians to consider this diagnosis when assessing patients with unexplained perioperative hemodynamic instability. Use of echocardiography proved to be an invaluable asset in the diagnostic process. If a diagnosis of TC is entertained, it is important to abstain...
from using inotropic therapy, which could further aggravate the cardiomyopathy.

**Supplementary materials**

Supplementary material associated with this article can be found, in the online version, at doi:10.1053/j.jvca.2021.02.022.

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