See Article page 111.

Commentary: Coronavirus disease 2019 in cardiac surgery: We are still learning and have yet to see all possible complications

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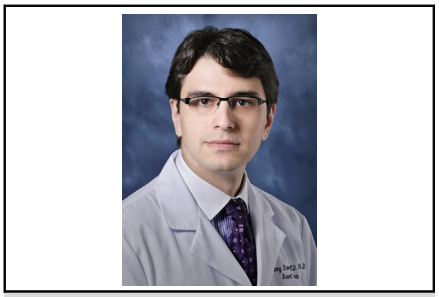

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CENTRAL MESSAGE

The hypercoagulable state of COVID-19 has been documented. This case report demonstrates an interesting complication of COVID-19 demonstrating that we still have more to learn about this virus.

A novel coronavirus outbreak identified in Wuhan, China, led to more than 48 million infected and nearly a million deaths.[1,2] Coronavirus disease 2019 (COVID-19) is characterized by extreme virulence and a complex spectrum of pathologies ranging in severity from mild to multiorgan failure[3] and death. Over the past 9 months, our knowledge of the pathophysiology and extrapulmonary manifestations of COVID-19 have grown tremendously. Despite this, we are constantly discovering new manifestations such as the development of a hypercoagulable state.[4-6] With an anxious global community focused on the development of a novel vaccine, it is easy to overlook that unique and potentially fatal complications are yet to be identified.

Manghat and colleagues[7] provide a single case report describing a newly discovered, potentially life-threatening complication. Specifically, acute aortic valve prosthesis thrombosis with resultant embolic myocardial infarction. The cardiothoracic surgery community has become accustomed to equating COVID-19 with pulmonary pathologies, acute respiratory distress syndrome, and hypercoagulable complications such as venothromboembolic disease. Venothromboembolic disease was recognized early during the pandemic with several institutions developing and recommending prophylactic anticoagulation strategies. Despite this knowledge, most patients are not prophylactically anticoagulated. The case presented by Manghat and colleagues[7] highlights the possible significant risk that can develop due to COVID–19-induced hypercoagulable state. To illustrate this point, Manghat and colleagues[7] describe the case of a 73-year-old woman with novel COVID–19-related complications; specifically, prosthetic aortic valve thrombosis shortly after aortic valve replacement. This resultant valve thrombosis led to coronary embolization and subsequent myocardial infarction. The finding suggests that with acute postoperative COVID-19 infection a significant hypercoagulable state may develop leading to significant morbidity. Readers might object, of course, and claim that no conclusive proof exists that this valve thrombosis resulted from COVID–19-induced hypercoagulability. But, in response, I assert the question: When was the last time we have seen a thrombosed aortic valve so early postoperatively and with embolic complications? Clearly, this is not a normal circumstantial paradigm. I argue that the COVID-19 infection was either the cause or played a significant, proximate causational role in the development of this complication. This case also highlights that the hypercoagulability associated with the COVID-19 infection may extend far beyond venothromboembolic disease and extend into arterial or implantable prosthesis thromboses. Our surgical community is rapidly realizing that the extrapulmonary manifestations of COVID-19 infection have yet to be fully elucidated, especially in the postcardiac surgery patients. Anticoagulation therapy falls into standard guidelines, with heparin followed by warfarin for thrombosed

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prosthetic valve. A new question now arises: Should we treat all COVID–19-positive cardiac surgery patients with warfarin? Such a sweeping strategy might seem overreaching, I suggest this treatment is prudent if we focus on prosthetic valve patients, in whom a tendency toward such complications has already been historically established.

The case report by Manghat and colleagues[7] is another in a series of revelations highlighting the rapid evolution of our knowledge of COVID-19, and reminding us that we still have a great deal more to learn about this landmark disease. Although no anticoagulation therapy after tissue valve implantation works in a majority of cases, it may not be appropriate in COVID-19 patients. Prophylactic anticoagulation when feasible—especially in a postoperative patient—would be a more appropriate and wiser choice in postvalvular replacement in COVID–19-positive patient. Although Manghat and colleagues[7] effectively treated their patient, the concern that I raise is the need for a national, standardized approach and treatment protocols for COVID-19 hypercoagulable states. But the most vital conclusion is that we are still learning and discovering new manifestations of COVID-19. If we lose sight of this valuable lesson, our patients may ultimately pay the price in morbidity and mortality. As the disease continues to proliferate across all continents, the report by Manghat and colleagues[7] is yet another reminder of the importance of cardiac surgery teams remaining hypervigilant in identifying novel, as well as known, manifestations and complications of COVID-19 before they become life-threatening.

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