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Searching for the reason why the results of infrarenal AAA open surgery are worsening

Pomy et al[1] compared the 30-day outcomes of open surgical repair (OSR) of infrarenal abdominal aortic aneurysms (IAAAs) from 2005 to 2010 and 2014 to 2019. They found that the outcomes were worse for both elective and emergency OSR in the latter period, despite the improvements in the preoperative assessments and advances in anesthesiology and intensive care techniques. In particular, they reported a significant increase in 30-day mortality for both the elective group (3.7% vs 3.2%; $P = .006$) and the ruptured cohort (41.4% vs 40%; $P < .0001$). Considering the limitations of a retrospective data review, their conclusions have raised important questions regarding the reasons for such a deterioration in the results.

The first consideration is that the worse outcomes could have been because in recent years many vascular surgeons have often opted for endovascular repair instead of performing OSR, not only for more anatomically complex AAAs, but also for standard IAAAs in patients fit for OSR. In addition, the recommendation of an endovascular first approach for ruptured IAAAs was determined from observational studies; however, randomized controlled trials did not conclude the superiority of endovascular repair.[2-5]

The second consideration is that small series can offer better results, such as a 30-day mortality rate of 12% in a series of 75 patients with ruptured IAAAs.[6] It could be interesting to investigate the reason for such discrepancies.

The last consideration is that the poor training of the newer generations of vascular surgeons for open surgical techniques is taking its toll. It has been documented that younger vascular surgeons have reduced skills in open vascular surgery. Thus, it is crucial to offer balanced training for two equally effective and valid techniques such as open and endovascular repair.

As claimed by the ancient philosopher Aristotle, the solution will probably be "in medio stat virtus" (virtue is found in the mean).

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From coronavirus disease 2019 to long coronavirus disease 2019 in vascular pathology

The interesting report by Faries et al[1] concerning the frequent complications in patients with coronavirus disease 2019 (COVID-19) who had been treated for arterial thrombosis merits some further discussion. SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), a multisystem disease, after an acute phase, can become protracted as prolonged COVID-19, lasting for 12 weeks, or for longer as long COVID-19. Medical treatments have often been unable to completely eradicate the viral load, neutralize all the negative effects, and restore the damaged physiologic function. In particular, COVID-19

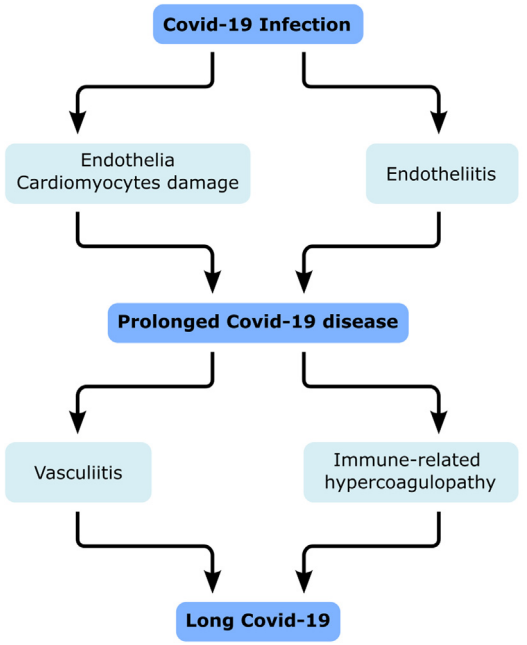

Fig. Flowchart showing pathophysiologic steps from coronavirus disease 2019 (COVID-19) to long COVID-19.

micro-organisms can penetrate into the arterial, venous, and capillary endothelia, causing accelerated pyroptosis and generating or aggravating corresponding endothelial dysfunction. In capillaries, the parietal infiltration of inflammatory cells will cause endotheliitis and subsequent microthromboses, with a large production of cytokines, typically interleukin-6.[2] Moreover, interrelating B and T lymphocytes will activate plasma cells to produce antibodies, autoantibodies, and complement anaphylatoxins (C3a and C5a), sometimes in a dysregulated manner, leading to a type 3 hypersensitive vasculitis and a more dangerous necrotizing leukocytoclastic vasculitis. An often associated secondary antiphospholipid-like syndrome, together with increased production of platelets from megakaryocytes, also present in the lung parenchyma, can generate an immune-correlated hypercoagulable state (Fig).[3-5] In medium- to large-size vessels, including coronary arteries, this results in an increased risk of thrombosis and, through a mechanism of platelet adhesion and deposition of inflammatory protein molecules, a rapid increase in atherosclerotic plaque formation. Aneurysms can increase in size owing to activated metalloproteinases, and typically in the aorta, can result in vasa vasorum thrombosis, increasing the risk of rupture.[6] Similarly, in cardiomyocytes, COVID-19 virions will accelerate their apoptosis, followed by myocardial invasion of inflammatory cells, typically lymphocytes. The consequent myocarditis favors the occurrence of dysrhythmia, decreases the ventricles' ejection fraction, and can lead to a secondary myocardial fibrosis. Moreover, the pulmonary lesions that can occur after COVID-19 owing to multiple areas of fibrosis can increase pulmonary resistance and the work of the right cardiac chambers.[7]

In conclusion, COVID-19 infection should be considered a disease not limited to an acute phase but with possible unpredictable long-term sequelae, correlating with a hyperregulated or dysregulated immune condition and a hypercoagulable state. Because of these factors, in addition to the often unknown power of the immune reaction of each patient, precise clinical studies should be performed before any decision for surgery, with close postoperative monitoring, especially for those with preexisting cardiovascular risk factors.

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