Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Conflict of Interest

None.

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Anesthetic Management of Patients Undergoing Aortic Dissection Repair With Suspected Severe Acute Respiratory Syndrome Coronavirus Disease 2019 (COVID-19) Infection

To the Editor:

We read with great interest the recent article on the anesthetic management of aortic dissection repair in patients with suspected severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. We would like to call your attention to our case, which sheds additional light on this topic. We report a case of a 50-year-old man admitted to the emergency department of A. Gemelli University Hospital in Rome, Italy, on March 11, 2020 for acute aortic dissection, who developed disseminated intravascular coagulation (DIC) in the immediate postoperative period. His symptomatology was characterized by syncope on the day before, without chest pain or dyspnea. Emergency computed tomographic scan showed a DeBakey II aortic dissection and pericardial effusion (Fig 1). No pulmonary alteration was observed. Afebrile at admission, he had traveled in Northern Italy, deemed to be at high risk for COVID-19 spread, and had a mild fever for the week before. Due to epidemiologic criteria, a nasopharyngeal swab was obtained for SARS-CoV-2 RNA test according to local protocol at the time, which ultimately was negative. The patient immediately underwent emergency surgery and all medical staff adopted third-level medical protection measures for COVID-19 safety procedures, including properly wearing disposable protective clothing, medical-grade masks such as N95, disposable surgical caps, medical-grade goggles, disposable latex gloves, and disposable shoe covers. The ascending aorta was replaced, requiring 20 minutes of hypothermic (28°C) circulatory arrest and selective brain perfusion. The patient was transferred to an isolation intensive care unit room and specific measures for infection containment were established despite the admission test being negative. In the intensive care unit, the patient presented massive bleeding from chest drainage. Rotational thromboelastometry (Tem GmbH, Germany) tests showed a hypercoagulable state, with secondary fibrinolysis, suggestive for a DIC condition like blood tests.[1] Coagulation derangement was treated and after 2 surgical revisions, without evidence of the surgical source of bleeding, a mediastinal packing was performed. Bleeding reduced substantially during the first postoperative day, and sternosynthesis was performed. Four days after surgery, the patient developed shock associated with multiorgan failure, fever, and increased Sequential Organ Failure Assessment score. Due to worsening of gas exchange and chest x-ray evidence of interstitial strengthening, a deep bronchoalveolar lavage (BAL) was performed for a second

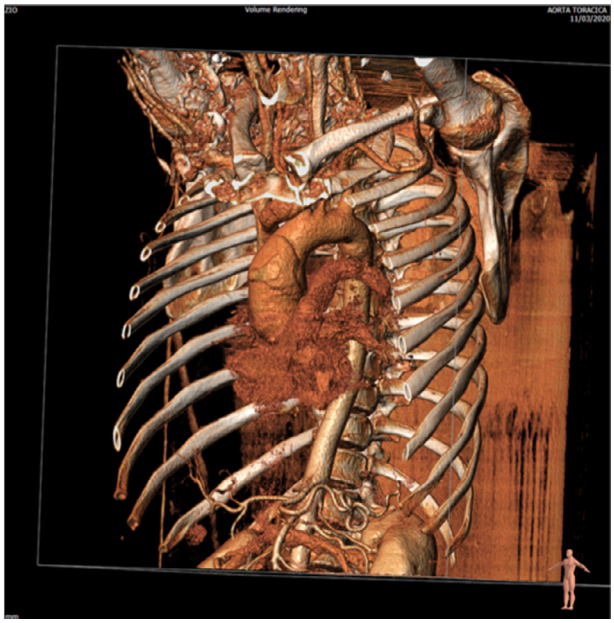

Fig 1. CT scan images. CT scan of the aorta shows 3-dimensional image reconstruction of thickened aortic wall originating from the sinotubular junction, ascending portion with a maximum diameter of 45 mm, without involvement of the epiaortic arterial branches. Without involvement of arch of aorta. Ascending aortic dissection and thickened aortic wall with pericardial and pericardial effusion. No pulmonary alteration in progress. CT, computed tomography.

SARS-CoV-2 RNA test, which turned out to be positive. The patient died after 10 days.

In our opinion, there are at least 2 aspects in this case that are worthy of being discussed. First, the presence of a high suspicion of COVID-19 is essential to adopt specific measures for infection containment, because false-negative results could hinder the prevention and control of the epidemic.[2] In a recent research letter, Wang et al showed that 93% of BAL swabs were positive in patients with confirmed SARS-CoV-2 compared to only 63% of nasal swabs,[3] probably due to the persistence in the lower respiratory tract. In our patient, it was essential to provide adequate personal protective equipment to healthcare professionals, and in a patient with suspected COVID-19–associated pneumonia, additional lower respiratory tract sampling was performed by trained and dedicated personnel according to World Health Organization guidelines.[4]

Second, the onset of immediate and massive bleeding associated with an overt DIC (International Society on Thrombosis and Haemostasis DIC score >5)[5] worsened the prognosis. The pathogenesis of DIC related to aortic dissection is associated with thrombus formation inside the dissected lumen and often is classified as an enhanced fibrinolytic process.[6] Disseminated intravascular coagulation can originate from and cause damage to the microvasculature, which can produce organ dysfunction if sufficiently severe.[5] In cardiac surgery, cardiopulmonary bypass often induces a systemic inflammatory response syndrome, which includes cascades of coagulation and a fibrinolytic condition.[7] A relation between poor prognosis and development of coagulopathy was described also in SARS-CoV-2 infection.[8-10] In a recent study, Tang et al[11] investigated the role of coagulopathy in severe novel coronavirus pneumonia. Indeed, they highlighted how DIC appeared in most of the deaths associated with a diagnosis of COVID-19. At the late stages of novel coronavirus pneumonia, the authors described how the levels of fibrin-related markers were elevated moderately or markedly in all deaths, which suggested coagulation activation and secondary hyperfibrinolysis condition. Nevertheless, coagulopathy often seems to be associated with pulmonary intravascular coagulation (PIC) and where DIC develops, it is restricted to late-stage disease.[12] SARS-CoV-2 may lead to an increase of intrapulmonary inflammation and PIC due to tropism for angiotensin-converting enzyme 2 receptor and its downregulation on type II pneumocytes.[13] But compared to DIC, during PIC, SARS-CoV-2–related platelet counts and fibrinogen concentration are not reduced substantially, despite marked increases in D-dimer concentration.[14] Autopsy series showed bilateral alveolar damage and fibrin thrombi within alveolar capillaries, with right ventricular dilatation as a possible effect of pulmonary hypertension,[15] associated with hemorrhagic necrosis.[16] Thrombosis mainly involves lungs but not other organs; hemorrhage is intrapulmonary and not generalized, and liver function is preserved.[17] In our patient, coagulation disorder seemed to be DIC- related, with generalized bleeding, persistent elevated D-dimer concentrations during 5 days after surgery, reduced platelet and fibrinogen values, and elevated liver function markers. After after surgery, we registered elevated cardiac enzyme concentrations and mild pulmonary hypertension, which does not exclude PIC but may be related to cardiac surgery. In conclusion, DIC, potentially SARS-CoV-2 infection- related, has preceded the pulmonary pattern, but the concomitant aortic dissection and cardiac surgery inflammatory response may have played a substantial role in triggering DIC, resulting in an overlap of severe pathologic conditions. According to our experience, we suggest using third-level medical protection measures for COVID-19 in emergency cardiac surgery until a definite diagnosis is made, considering that BAL is a more specific and sensible test than nasopharyngeal swab in intubated patients. We also suggest that SARS-CoV-2 infection may play a role in exacerbating and potentiating the inflammatory response and coagulopathy in aortic dissection surgery, resulting in potentially greater risk of DIC and bleeding in the postoperative period.

Conflict of Interest

None.

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What Is the Optimal Heart Rate Control for Atrial Tachyarrhythmia Following Cardiac Surgery?

To the Editor:

I congratulate Khan and colleagues for their successful heart rate reduction therapy using ivabradine in 7 pediatric patients with postoperative atrial tachyarrhythmia following cardiac surgery.[1] There are several concerns about the implication of their findings.

The authors administered ivabradine via the gastric tube or orally in patients refractory to amiodarone therapy. Recently, the ultra-short-acting beta-blocker landiolol has been used to decrease heart rate in patients with atrial tachyarrhythmia.[2] Intravenous administration of landiolol can normalize atrial tachyarrhythmia within hours. The authors reported 1.8 hours as the average time of initial response to ivabradine, which was defined as the time to reduction of heart rate by at least 20 beats per minute.[1] Earlier normalization of atrial tachyarrhythmia would prevent more hemodynamic deterioration.

Ivabradine was discontinued in all patients before discharge.[1] Long-term optimization of heart rate seemed to be associated with improved prognosis in the heart failure cohort.[3] Our team recently proposed optimization of heart rate using echocardiographic transmitral flow pulsed Doppler procedure.[4] In short, both the E wave and A wave should be adjacent, without any overlap, to maximize cardiac output. Clinical implication of short-term or long-term ivabradine merits future studies.

Declaration of Competing Interest

None.

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Simple and Reversible Venoarterial Extracorporeal Membrane Oxygenation Wean-Off Simulation Using Inflow-Outflow Bridging

To the Editor:

WITH GREAT INTEREST, we read the review article by Hoyler et al. regarding the management of venoarterial extracorporeal membrane oxygenation (VA-ECMO).[1] An often underappreciated, but highly important, step in the management of patients supported by VA-ECMO involves proper timing of support discontinuation, as is mentioned. However, in the algorithm they describe, the patient is not fully separated from VA-ECMO until the cannulae are withdrawn. Thus, true hemodynamics without VA-ECMO remain unknown. Early decannulation may lead to cardiopulmonary decompensation and VA-ECMO reinitiation, and unnecessarily prolonged VA-ECMO support may increase the risk of complication, including physical function deterioration. We present a simple and reversible VA-ECMO wean-off simulation that uncouples the patient from VA-ECMO support for an extended period without the need for decannulation while also maintaining flow in the VA-ECMO circuit. The simulation can be promptly reversed if the patient cannot be weaned successfully.

Patients who can tolerate 1.0 L/min or less flow, with minimal inotrope/pressor, are taken to the operating room. Cardiac function and hemodynamics are assessed via transesophageal echocardiogram, jugular venous catheterization, and blood gas (ABG) analysis. Typically, cardiac index greater than 2.2 L/m/m^2, with mild- to- moderate inotrope/vasopressor support, is required to successfully wean from VA-ECMO. A heparin bolus is then infused to maintain an activated clot time (ACT) of greater than 300 seconds. Flow is slowly reduced to 0.5 L/min; 2 Y-shaped connectors are then used to create a bridge between the inflow and outflow cannulae. The outflow cannula distal to the bridge is then clamped. Only the outflow cannula is clamped because the VA-ECMO circuit is closed. Flow through the VA-ECMO circuit is maintained through the bridge (Fig 1, A and B). The outflow cannula clamp is moved to the bridge for 15 seconds every 5 minutes to flush the distal cannulae by allowing blood to flow through the native VA-ECMO circuit, thereby preventing clot formation (Fig 1, C and D). After 30 minutes of simulation, hemodynamics and respiratory status are examined via evaluation of cardiac index, mixed venous oxygen saturation, and arterial blood gases.

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