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team decided to perform crash bypass if the hemodynamics worsened; however, over the next 5 -to -10 minutes, saturation increased and the ET CO_2 level took almost 15- to -20 minutes to reach its baseline value (see Fig 1). Surgery was resumed after 20 minutes, and the rest of the course was unremarkable. The patient experienced a smooth postoperative course, with no neurologic deficits and was discharged on day 4 after surgery.

The CO_2 blower is used extensively and is effective in sustaining a bloodless field during OPCAB. The principal cause of PE in this case was that the surgeon used a CO_2 blower during repair of the injured RA, which is a low-pressure chamber, which must have pushed the gas into the right side of the heart and subsequently into pulmonary artery. There are a small number of reports of a CO_2 gas blower causing PE. Hirata et al.⁶ reported CO_2 embolism via a torn right ventricular outflow tract during LAD grafting in OPCAB that was caused by a CO_2 blower. Lee et al.⁴ also reported a massive CO_2 embolism via a torn coronary vein during OPCAB that was caused by a CO_2 blower. Even though we did not have any objective evidence to demonstrate gas bubbles in the right side of the heart using transesophageal echocardiography (TEE), the surgical findings and the associated clinical picture made us believe that the CO_2 gas blower was the underlying cause of the PE. The central venous pressure was 10- to 12- mmHg during the anastomosis, and it seems unlikely that air was sucked into the venous system from the atmosphere. Because the patient already was in the Trendelenburg and left lateral positions for RCA grafting during the event, the patient may easily have from the insult. Sustained hemodynamic derangement necessitates institution of cardiopulmonary bypass as reported by Chang et al,⁶ who successfully managed a massive PE with cardiac arrest using cardiopulmonary bypass. A pig model showed that TEE is the “gold standard” monitor, with earlier detection of gas emboli than ET CO_2 change. Because CO_2 is highly soluble in blood and swiftly absorbed from the bloodstream, there is also a time lag between the TEE image and ET CO_2 change during acute CO_2 embolism. To conclude, a CO_2 blower should not be used for repair of venous systems in order to avoid CO_2 embolism.⁵ Careful surveillance with TEE may be considered for early detection of a gas embolism, and ET CO_2 is an invaluable tool in the detection of PE clinically if TEE is unavailable, as in our case.

Conflict of Interest

None.

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COVID-19 With Limb Ischemic Necrosis



To the Editor:

A 53-year-old man from Wenzhou, Zhejiang, China was admitted to the First Affiliated Hospital of Wenzhou Medical University on February 1, 2020, with a 9-day history of fever with dizziness, cough, and sputum. On presentation, his temperature was 38.6°C. Laboratory tests showed a C-reactive protein concentration of 70.5 mg/L (normal range, 0.00-10.00 mg/L). Complete blood count showed elevated white blood cell count ($14.9 \times 10^9/\text{L}$ [normal range, $3.5-9.5 \times 10^9/\text{L}$]), neutrophils ($14.2 \times 10^9/\text{L}$ [$1.8-6.3 \times 10^9/\text{L}$]), and monocytes ($0.42 \times 10^9/\text{L}$ [$0.1-0.6 \times 10^9/\text{L}$]). D-dimer was 0.68 mg/L (0.00-0.5 mg/L) and platelet count was $163 \times 10^9/\text{L}$, whereas the lymphocyte count (0.25) decreased (normal range, $1.0-3.2 \times 10^9/\text{L}$). The computed tomography scan presented multiple ground-glass opacities with consolidation and bilateral lung involvement. The patient was diagnosed with coronavirus disease 2019 (COVID-19) on the basis of reverse transcription-polymerase chain reaction (RT-PCR) analysis of sputum samples. The patient developed complications including septic shock, multiple organ dysfunction syndrome, acute respiratory distress syndrome, thrombocytopenia, acute kidney injury, and catheter-related infections. The patient was treated with antibacterial, antiviral, and corticosteroid treatments, mechanical ventilation, and extracorporeal membrane oxygenation.

During hospitalization, peripheral ischemia appeared on the right fingers and toes and gradually resulted in gangrene (Figs. 1 and 2). Beginning on February 29th, the patient's right fingertip began to show a slight cyanosis, and on March 7th, the patient's right fingertip cyanosis continued to progress and the boundaries were clear (Fig 1). After March 10th, the patient started to develop black gangrene-like manifestations on his fingertips (Fig 2).

Limb ischemic necrosis is a rare but dreadful complication. Tissue necrosis in the fingers was irreversible. Although the precise mechanism of limb ischemic necrosis is unclear, this



Fig 1. Right fingers showed cyanosis.



Fig 2. Black gangrene-like manifestations on the fingertips.

may be related to high-dose use of norepinephrine.¹ Acute ischemic hepatitis (“shock liver”) has been identified as a potential risk factor for limb ischemic necrosis or symmetrical peripheral gangrene.² The previous study shows that abnormal coagulation results, especially markedly elevated D-dimer and fibrin degradation product, are common in deaths with COVID-19.³ Therefore, the occurrence of limb ischemic necrosis in COVID-19 patients should be monitored closely.

Conflict of Interest

The authors declare they have no conflict of interest.

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How 3D Printing Can Prevent Spread of COVID-19 Among Healthcare Professionals During Times of Critical Shortage of Protective Personal Equipment



To the Editor

Over the last several months, severe acute respiratory syndrome coronavirus 2 (COVID-19) has continued to spread all over the globe at an alarming rate, with total disregard for patients’ pre-existing medical conditions, age, or other demographics.¹ The mortality rate is substantially higher than influenza, and the death rate in the United States seems to be surpassing that of both China and Europe.² Although healthcare professionals report for duty each morning and evening to care for an increasing number of patients with various gradations of disease, their own safety and health remain threatened. The medical community has experienced a sobering reality: there is an utter lack of resources for patients as well as for providers. In particular, personal protective equipment (PPE) is at a critically low supply, which puts patients and providers at unacceptable risk. In the 2002 coronavirus severe acute respiratory syndrome outbreak, healthcare workers comprised an alarming 21% of cases, a trend we aim to prevent.³ Many strategies have been implemented by various individuals and companies to meet these needs, or to improvise in the meantime.⁴ Without the equipment necessary to protect healthcare workers, not only is there significant disruption of hospital work and unobtainable pressure put on the supply chain, but there is also profound risk to the lives of the people meant to care for the rest of society.

The spread of COVID-19 is mediated by various contact with droplets as well as direct airborne exposure.⁵ Varying degrees of protection are necessary for a range of procedures and patient interactions.⁶ At a minimum, any provider taking care of a patient with suspected or proven infection must wear a gown, gloves, an N95 respirator, and an eye shield/facemask. Healthcare providers engaged in procedures involving the nose, mouth, or airway need even more aggressive protection from airborne viral particles, in the form of powered air-purifying respirators, or similar.³

There are a number of ways in which the healthcare professional may be exposed to viral particles, and therefore risk personal infection or infection of other patients. Proper PPE therefore is not a luxury but a requirement to prevent mass infection and continued spread. The equipment available at this time is insufficient for a number of reasons, but first and