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.

Table 1 Detailed Medication Plan of Immunosuppression and Changes in Existing Medication

Medication	Dose	Baseline Day 0	Midpoint Day 10	Discharge Day 21
Tacrolimus	mg/day orally	4	5	6
Prednisolone	mg/day orally	5	5	5
Trimethoprim/sulfamethoxazole	mg/day orally/twice weekly	960	960	960
Metamizole	mg/day orally	—	500	—
Salbutamol	inhalation dose 4 times/day	—	100 μg	100 μg
Meropenem	g/day intravenously	4	4	—

RNA, thereby establishing a diagnosis of COVID-19. She was hospitalized, and a chest X-ray showed chronic postoperative dystelectasis on the left side with some increase in density (Figure 1) compared with December 2019. A chest computerized tomography scan showed ground glass opacities mainly in the left lower lobe with left-sided parenchymal consolidation or partial atelectasis (Figure 2). Oxygen supplementation with 1 to 2 liters/minute was administered. Antibacterial therapy was empirically started based on suspected bacterial superinfection. Otherwise, no major changes in medication were performed. A detailed medication plan is shown in Table 1. She remained asymptomatic and remained stable. RT-PCR testing was performed on a weekly basis and remained positive on Day 7 and Day 14. Cycle threshold levels of SARS-CoV-2 E- and S-gene are provided in Table 2. Cycle threshold values increased at every sample time point, indicating a decrease in virus levels over time. On nasopharyngeal swabs on Day 21, no SARS-CoV-2-RNA could be detected by RT-PCR. No oxygen was required from Day 17 until discharge. The patient was discharged home on Day 21.

In a report based on data collected in China from 1,099 patients during the 2 first months of COVID-19 outbreaks, 5% of the patients were admitted to the intensive care unit, 2.3% underwent invasive mechanical ventilation, and 1.4% died.[2] Although older patients (>50 years old) and patients presenting with coexisting disorders are more prone to suffer from severe disease, data on disease presentation and evolution in immunocompromised patients are scarce. In the first report of the COVID-19 outbreak, 2 patients (0.2%) with COVID-19 and a not-otherwise-specified immunodeficiency were reported. Both patients had nonsevere disease, and neither was admitted to the intensive care unit, underwent invasive mechanical ventilation, or died. In a 52-year-old patient who had kidney transplantation 12 years earlier and confirmed COVID-19, successful recovery was achieved following reduction of immunosuppressant therapy coupled with low-dose methylprednisolone-based therapy.[3] In 2 heart transplant recipients, both recovered after supportive therapy with antibiotics and antiviral therapy coupled to reduction of immunosuppressive therapy.[4] From these first very early experiences with COVID-19 in renal, heart, and lung transplant recipients, disease presentation seemed to be similar to the general population. Whether COVID-19 is more severe or probably mitigated owing to the effects of the immunosuppression on virus replication in patients after solid organ transplantation is still unknown, but recovery was so far possible in most reported cases, although some anecdotal unpublished reports from Italy suggest a higher morbidity in older transplant recipients. Based on this experience, a higher clinical suspicion is warranted, and early testing is recommended, because COVID-19 can be present even in relatively asymptomatic patients after lung transplantation.

Table 2 Ct Levels in SARS-CoV-2 RT-PCR

Date	Ct E-gene	Ct S-gene
Day 0	23.59	22.07
Day 7	34.34	33.96
Day 14	35.23	35.28
Day 21	target not detected (≥40)	target not detected (≥40)

Abbreviations: Ct, cycle threshold; RT-PCR, reverse transcriptase—PCR; SARS-CoV-2, severe acute respiratory syndrome—coronavirus 2.
Ct levels ranged from 20 to 39.

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Donor organ evaluation in the era of coronavirus disease 2019: A case of nosocomial infection

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A 45-year-old man with a history of substance abuse presented with altered mental status secondarily to a fall.

Computed tomography (CT) of the head suggested anoxic brain injury with no hemorrhage. Chest CT angiography was consistent with a right lower lobe pulmonary artery segmental embolus. On Day 2, he was found to have a large, acute left middle cerebral artery ischemic infarct. Subsequently, on Day 7, he developed sub-falcine herniation. He remained afebrile, but his white blood cell count increased from 7,600 to 17,800 cells/mm^3 of blood with no bacterial infection on respiratory and blood cultures. During organ-donation work-up, a repeat chest CT (on Day 8) showed resolution of his pulmonary embolus, but new scattered, bilateral ground-glass opacifications were noted, which prompted bronchoalveolar lavage and nasal swab specimens for coronavirus disease 2019 (COVID-19) testing. These were positive 24 hours later, and the patient was declined as an organ donor. This case raises several points regarding the assessment of donors for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. Given the frequent absence of donor history, the extent of community spread, concern for the risk of nosocomial acquisition, and concomitant hazard to medical personnel, donor screening should be mandatory. The International Society for Heart and Lung Transplantation currently recommends that all donors should be tested for SARS-CoV-2 infection *if testing is available*.[1] Early screening may be prudent, particularly when medical history is sparse or absent. Admission to a facility or units known to have cases of COVID-19 increases concern for nosocomial transmission. In this case, the patient may have arrived with pre-existing asymptomatic COVID-19 infection but may also have acquired the infection after arrival at the donor hospital.[2,3] In such cases, even if an initial screening test is negative, a repeat test should be performed before the recovery of organs. For organ procurement organizations, tests may need to be sent out if the donor hospital is unable to provide in-house testing, resulting in significant delays in allocation. Although reverse transcriptase polymerase chain reaction (RT-PCR) sensitivity varies widely by sampling site, concurrent evaluation of chest CT seems to greatly increase sensitivity for the disease.[4] In this candidate donor, the final chest CT demonstrated the development of bilateral ground-glass opacities that were consistent with COVID-19, which was confirmed by respiratory RT-PCR. The chest CT was convincing evidence to defer making a decision to accept the organ until the RT-PCR result was available. Multimodality RT-PCR testing should also be considered as the virus may be present in mucosal areas such as the gut. However, availability of such testing may be limited. Although the disease primarily affects the lungs, it is not clear whether other organs may be safely transplanted. From a cardiac standpoint, the angiotensin-converting enzyme 2 receptor is required for SARS-CoV-2 entry and is expressed on cardiac myocytes. There has been evidence of fulminant myocarditis in COVID-19, and troponin elevation has been associated with increased mortality. Because the outcomes of transplanting organs from a COVID-19–positive donor and the extent of cardiac involvement in COVID-19 are currently unknown, it is our practice at the time of publication to decline organs from donors with positive RT-PCR testing, even for an isolated cardiac transplant. Other considerations include a significant risk of transmission to the procurement team, the lack of resource-effective surveillance strategies for donor transmission, absence of proven treatments for this potentially lethal condition, and potential for turning the recipient into a vector for viral transmission. Owing to the many uncertainties and rapidly evolving data regarding SARS-CoV-2, it is vital to develop donor testing protocols for COVID-19 during this pandemic.

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COVID-19 pneumonia in a dual heart–kidney recipient

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A 61-year-old African American man with a history of hypertension, coronary artery disease, end-stage renal disease (on hemodialysis since 2014), and end-stage heart failure secondary to arrhythmogenic right ventricular cardiomyopathy underwent dual organ heart–kidney transplantation in May 2019. Approximately 2 months after transplant, mycophenolate was discontinued owing to episodes of pancreatitis, leukopenia, and detectable BK polyomavirus. Since then, the patient was maintained on tacrolimus (goal level of 8 ng/ml) and low-dose prednisone (5 mg/day). Eight months after transplant, the patient developed a mild influenza A infection that was treated with oseltamivir for 10 days. After 6 weeks, approximately 10 months after heart–kidney transplantation, the patient re-presented with cough productive of yellow sputum for 3 days, associated with pleuritic chest pain, dyspnea, nasal congestion, and subjective fevers. He denied travel or exposure to known individuals infected with coronavirus disease 2019 (COVID-19). Initial vital signs were within normal limits and physical examination was unremarkable. The blood oxygen saturation on room air was 96%. Respiratory viral panel was negative, and white blood cell count and blood lactate were normal. Absolute lymphocyte count was