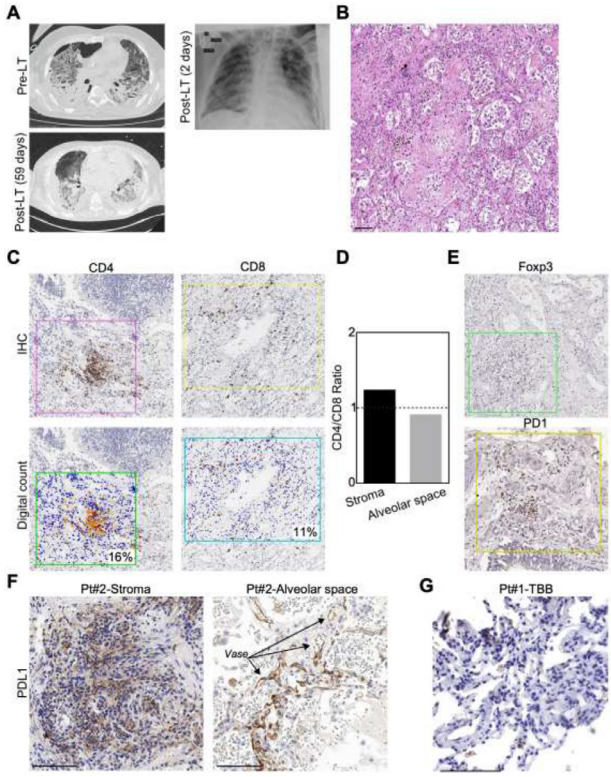
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

second patient (Pt#2) died because of allograft rejection at day 62 post LT and explanted lungs were retrieved. CT imaging of the lungs was performed three days before death. Morphological examination was performed by H&E, whereas the immunophenotyping was performed by immunohistochemistry.
Results: Imaging and morphological examination of Pt#2 lungs indicated the presence of a graft dysfunction with features of a restrictive, widespread usual interstitial pneumonia-like syndrome (Fig. 1A, B). The immunophenotyping showed that B-lymphocytes (CD20-positive) were nearly absent, CD8-T-cells were not particularly expanded (mean positive cells within the lung stroma=13.8%; Fig. 1C), and the CD4/CD8 ratio was not decreased (Fig. 1D). The T-regs (Foxp3-positive) were 6% of the overall population (Fig. 1E). Analysis of the immune checkpoint molecules PD1, Tigit, CTLA4 and PDL1 showed that the expression of PD-L1 alone was highly increased in vases and in alveolar cells of rejected lungs, whereas it was nearly undetectable in the TBB from Pt#1 (Fig. 1F, G).
Conclusion: PDL1 expression in vases was previously documented as a sign of indirect ARDS. Together with our preliminary data, we can hypothesize that PDL1 may play a role in tissue effacement and graft failure, possibly indicating poor allograft prognosis.



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Immunosuppressive Treatment Does Not Prevent Humoral and Cellular Virus-Specific Immunity in Heart or Lung Recipients with SARS-CoV-2 Pneumonia

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Purpose: Clinical characteristics of SARS-CoV-2 infection and virus-specific humoral and cellular response were analyzed in 4 heart (HR) and 3 lung (LR) Tx recipients in standard triple immunosuppressive regimen.
Methods: SARS-CoV-2 infection was diagnosed by real-time PCR on nasopharingeal swabs (NPS). T-cell response to structural antigens Spike (S), Envelope (E), Membrane (M) and Nucleocapsid (N) was evaluated by PBMC stimulation with overlapping peptides spanning the entire viral proteins and subsequent detection of cell activation markers CD137 and CD25. Serum IgG antibody to S and N, and IgA antibody to S were determined by ELISA.
Results: Three patients developed SARS-CoV-2 infection early (<3 months) and four patients late (>3 years) after Tx. One HR was asymptomatic, one LR presented only gastrointestinal symptoms, and five patients developed dyspnea with radiologic signs of interstitial pneumonia (in one HR ICU admittance was necessary. All patients recovered from SARS-CoV-2 infection, with viral clearance from NPS within 3 weeks. However, two HR (one early and one late HR) died at 6 and 4 months after infection because of multi-organ failure and sudden death. Both deaths were considered as unrelated to SARS-CoV2 infection. Patients who had no lung involvement did not develop specific antibody response, while all the other five patients developed IgG and IgA antibodies to S, and IgG antibody to N, within 2 months after infection. All the symptomatic patients developed a detectable CD4+ T-cell response to two or more antigens. Four patients were subsequently examined >3 months after infection, showing the persistence of IgG and IgA antibodies to S and a decline of IgG antibody to N, while CD4+ T-cell response to N was maintained. Timing of Tx did not affect the occurrence of virus specific immunity.
Conclusion: Although this series is small, the data indicate that immunosuppression does not prevent the development of a specific humoral and cellular anti-SARS-CoV-2 response, more likely in patients who have experienced clinically relevant pneumonia. These preliminary data encourage the maintenance of regular follow-up to monitor the persistence of immune response and the potential occurrence of SARS-CoV-2-related sequelae in heart and lung recipients.

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Rapid ECMO Training for Nurses in Response to the COVID-19 Pandemic

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Purpose: From March 17th to April 29th, our ECMO Program placed 30 adult patients on venovenous extracorporeal membrane oxygenation (VV-ECMO) for management of coronavirus disease 2019 (COVID-19). This acute increase in volume placed a strain on our available ECMO-competent nursing staff. Although perfusionists function as our ECMO specialists, our critical care nurses provide continuous circuit monitoring and respond to emergencies. Because of the need to increase the number of ECMO-competent nurses, on March 27th a focused, two-hour COVID-ECMO training course was implemented.
Methods: We retrospectively reviewed the number of ECMO care hours provided by our nursing staff and separated the nursing staff into two cohorts. Group A consisted of nurses who had undergone ECMO training prior to the pandemic (n=126). Group B consisted of nurses whose initial ECMO training occurred during the pandemic (N=145). We then compared the number of nursing hours provided by each cohort before and after training.
Results: From March 27th to May 4th, 145 nurses completed training, increasing our total number of ECMO-competent nurses from 126 to 271 (115% increase). From March 17th to June 30th, 20,677 ECMO care hours were provided. Pre-training, all 634 care hours were 100% provided by Group A nurses. Post-training, 20,043 care hours were provided, consisting of 39% Group A nursing coverage and 61% Group B nursing coverage. There were no differences in nursing related ECMO-emergencies between the two groups. At the conclusion of the surge, 28 out of 30 (93%) patients survived ECMO and 26 out of 29 patients (90%) survived to hospital discharge. One patient has a pending hospital disposition.
Conclusion: Rapid implementation of an abbreviated ECMO education program for nurses is feasible. It met the time-sensitive needs of the COVID-19 pandemic and provided safe nursing coverage to patients requiring ECMO.