



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.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

Pathophysiology of COVID-19-associated acute respiratory distress syndrome

Compared with diseases from other coronaviruses (ie, severe acute respiratory syndrome and Middle East respiratory syndrome), COVID-19 has more adverse effects on the cardiovascular system, leading to a high incidence of cardiovascular events—most notably life-threatening pulmonary vessel injury and cardiac dysfunction, with and without severe myocardial injury.¹ Small pulmonary vessel injuries and thrombosis associated with pulmonary blood flow alterations followed by right heart dilation and right ventricular failure have been found among the major causes of death related to COVID-19.¹

In *The Lancet Respiratory Medicine*, Giacomo Grasselli and colleagues² showed that COVID-19-associated pulmonary injury with acute respiratory distress syndrome (ARDS) is characterised by decreased pulmonary compliance and increased lung weight. Of particular importance is the observation that, when pulmonary damage occurred together with high D-dimer concentrations,

mortality increased: 28-day mortality was twice as high in the high D-dimers–low compliance patient group (40 [56%] of 71 patients) compared with the low D-dimers–high compliance group (18 [27%] of 67 patients). CT also revealed filling defects or occlusions of the pulmonary vasculature that were more prominent in patients with high D-dimer concentrations (15 [94%] of 16 patients with elevated D-dimer concentrations had bilateral, diffuse areas of hypoperfusion, consistent with the presence of thrombi or emboli). These observations strongly indicate that pulmonary vascular thrombosis can be the main cause of COVID-19-related death. In this regard, it would be particularly important to have information on pulmonary arterial pressure and right-sided heart anatomy and function because, due to the direct alterations and damages of the pulmonary circulation, the pathophysiological role of the heart–lung interactions will become mainly involved in the development and progression of life-threatening right ventricular dysfunction.

Echocardiography has revealed elevated pulmonary arterial pressure in nearly 70% of patients with COVID-19.³ In one study, right ventricular dilation

was present in 31% of hospitalised patients and on multivariate analysis, right ventricular dilation was the only variable associated with mortality.⁴ In heart-transplanted patients with COVID-19 who needed mechanical ventilation, the high mortality (up to 87%) appeared mainly associated with increased pulmonary arterial pressure and right ventricular dysfunction, whereas no patient showed left ventricular dysfunction.⁵

Putting together all the information regarding D-dimers, respiratory system compliance, pulmonary hypoperfusion, pulmonary arterial pressure, and right-sided heart dilation and dysfunction leads to the conclusion that these aspects, and particularly the often neglected right ventricular, need more attention. Hand-held laptop-based echocardiography devices, which can be sufficient for assessment of right-sided heart alterations and dysfunction, could substantially improve the management of patients with symptomatic COVID-19 to reduce the particularly high mortality rates.

I declare no competing interests.

Michael Dandel
mdandel@aol.com

German Centre for Heart and Circulatory Research (DZHK) Partner Site Berlin, Berlin 107085, Germany

- 1 Gupta A, Madhavan MV, Sehgal K, et al. Extrapulmonary manifestations of COVID-19. *Nat Med* 2020; **26**: 1017–32.
- 2 Grasselli G, Tonetti T, Protti A, et al. Pathophysiology of COVID-19-associated acute respiratory distress syndrome: a multicentre prospective observational study. *Lancet Respir Med* 2020; **8**: 1201–08.
- 3 García-Cruz E, Manzur-Sandoval D, Rascón-Sabido R, et al. Critical care ultrasonography during COVID-19 pandemic: the ORACLE protocol. *Echocardiography* 2020; published online Aug 29. <https://doi.org/10.1111/echo.14837>.
- 4 Argulian E, Sud K, Vogel B, et al. Right ventricular dilation in hospitalized patients with COVID-19 infection. *JACC Cardiovasc Imaging* 2020; published online May 15. <https://doi.org/10.1016/j.jcmg.2020.05.010>.
- 5 Rivinius R, Kaya Z, Schramm R, et al. COVID-19 among heart transplant recipients in Germany: a multicenter survey. *Clin Res Cardiol* 2020; published online Aug 11. <https://doi.org/10.1007/s00392-020-01722-w>.



Published Online
November 13, 2020
[https://doi.org/10.1016/S2213-2600\(20\)30507-5](https://doi.org/10.1016/S2213-2600(20)30507-5)

