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Intensive Care Unit Strain and Mortality Risk Among Critically Ill Patients With COVID-19—There Is No "Me" in COVID

Lewis Rubinson, MD, PhD

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The coronavirus disease 2019 (COVID-19) pandemic has affected numerous communities, and reports of overburdened hospitals, specifically critical care units, have become commonplace. High-quality supportive care remains the foundation for ensuring that people with COVID-19 who are critically ill have the best chance of surviving. Such care in prepandemic times relied on sufficient expert staffing, specialized equipment, and appropriate environments of care to reliably implement a myriad of processes that are associated with better outcomes. Given that these resources may not all be consistently available when severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is rapidly spreading throughout a community, Bravata et al[1] sought to evaluate the association of critical care strain from March through November 2020 with COVID-19 mortality in 88 Veterans Administration hospitals with 10 or more intensive care unit (ICU) beds in the US.

The authors used 2 metrics for critical care strain: ICU load (ie, the ratio of the mean number of patients with COVID-19 in the ICU during the patients' hospitalization and the total number of ICU beds at the facility) and ICU demand (ie, the ratio of the mean number of patients with COVID-19 in the ICU and the peak number of patients with COVID-19 in the ICU at that facility during the study period). Patients with COVID-19 treated in the ICU during periods of high ICU load or demand fared worse than those treated during times of low COVID-19 ICU load or demand. Being elderly and requiring mechanical ventilation had a stronger association with hazard of death, but ICU strain had a clear association with mortality. If these results represent true causality, they provide additional support for public health strategies to "flatten the curve."[2]

This important early investigation suggests that mortality for patients with COVID-19 who are critically ill may be associated with the extent of burden of other patients with COVID-19 in the ICU. Additionally, the timing of an individual's hospitalization within the surge of the virus across the community may be associated with ICU survival, again as a result of the concurrent critical care COVID-19 burden during the individual's admission. Given these findings, it is appealing to advocate for interventions to reduce ICU load and ICU demand, because many patient factors associated with increased COVID-19 mortality may not be easily modified (eg, age and comorbidities). Some jurisdictional authorities have used ICU load thresholds to trigger nonpharmacologic interventions for reducing the pace of community transmission, and this should be broadly considered. It may be more difficult to operationalize ICU demand, because the peak COVID-19 ICU census can be gleaned only retrospectively. For ICU load mitigation, the authors suggested a regional approach to COVID-19 critical care patient redistribution. These data may provide support for such a tactic, but the challenges of moving severely ill patients among facilities must not be undertaken lightly. While interfacility transport of individuals with COVID-19 who are critically ill has clearly been done safely, each transport has significant risk. Therefore, the redistribution system, if undertaken, must be well designed and implemented to ensure that the solution is not associated with new adverse events.

In this cohort, most strain due to ICU load occurred earlier in the outbreak. Because high levels of ICU load were not seen later in this study, the association with mortality may be partially or even entirely explained by secular trends in care. Early in the pandemic, a number of unproven therapies were being used for potential antiviral effects or immunomodulation. The Recovery trial[3] results regarding dexamethasone and the emergency use authorization access to remdesivir[4] both arrived later during this study period, and a number of potentially harmful therapies (eg, hydroxychloroquine

with or without azithromycin[5]) may have been used less frequently as the outbreak matured. Additionally, a number of potentially beneficial treatment strategies may have been adopted over time (eg, anticoagulation strategies[6] and use of permissive hypoxemia when appropriate). Caution is therefore prudent prior to using this study in isolation to influence response policy, as the analyses did not include patient-level treatments for multivariable adjustment.

Demand in the ICU was also associated with mortality for patients with COVID-19 who were critically ill. High ICU demand had less skewing toward earlier months compared with ICU load, and thus secular changes are less likely to explain this association. However, there probably was still a learning curve for the hospitals that were not affected until later in the outbreak, so unmeasured variations in treatments may still explain some of the association. In addition, the increased COVID-19 ICU mortality during peak surges in the community may be associated with the harvesting effect, in which individuals who are most likely to die become ill early in a specific community's outbreak. If the harvesting effect overlaps with the peak surge in the community, then the excess mortality can appear to be associated with ICU demand but in fact be associated with patient factors not adjusted for in the model. The authors adjusted for some patient factors, including age, comorbidities, and Acute Physiology, Age, Chronic Health Evaluation (APACHE) score; still, it is possible that the patient population was different over time, and this difference could be associated with residual confounding in the multivariable model. Lastly, patients who were admitted to ICUs could have been less ill when there was less ICU demand from COVID-19 patients. If the criteria for ICU admission selected for patients with less severe illness during periods of low ICU demand and ICU load, then increased mortality could have been associated with patient selection rather than ICU demand.

Despite these uncertainties, high levels of ICU load and ICU demand could be causative factors associated with excess mortality. If this was the case, one would expect that processes of care associated with critical care mortality (eg, staffing ratios, processes to reduce nosocomial infections and availability of experts in mechanical ventilation) would be different at different levels of ICU strain. Studies that address process in addition to structure and outcome are essential to validate the findings of Bravata et al.[1] Finally, while the authors should be commended for using a convenience sample from readily available data from the US Veterans Administration, these hospitals are quite different from nonfederal hospitals, and the impact of ICU strain in other hospitals should be investigated.

In summary, Bravata et al[1] present important evidence that survival for patients with COVID-19 in the ICU may be associated with the number of other patients with COVID-19 who are concurrently in the same ICU. If so, measures to flatten the curve and redistribute individuals with COVID-19 who are critically ill to other less impacted hospitals may be important strategies for improving survival. In light of the important policy implications, additional analyses are urgently needed to investigate whether this association is causal. If causality is supported, delineation of which care processes are suboptimally provided as ICU load and demand increase will be important to assist hospitals to buttress these processes in hopes of reducing the impact of ICU strain on mortality.

ARTICLE INFORMATION

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Corresponding Author: Lewis Rubinson, MD, PhD, Morristown Medical Center, Atlantic Health System, 100 Madison Ave, Box 20, Morristown, NJ 07960 (lewis.rubinson@atlantichealth.org).

Author Affiliation: Morristown Medical Center, Atlantic Health System, Morristown, New Jersey.

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