Pulse Oximetry as a Biomarker for Early Identification and Hospitalization of COVID-19 Pneumonia

The word “emergency” is defined by time. We work in time-dependent windows of diagnosis and treatment. Consider the enormous impact of biomarkers and imaging now standard in the ED (troponin, D-dimer, and lactic acid and lightning-fast pan-scanning CT and bedside ultrasound). Faster detection of critical illness has also led to more ED administration of pharmacologic interventions (aspirin, thrombolytics, antibiotics, fluid resuscitation, pressors, TXA, four-factor PCC, etc.). Helicopter transport, intraosseous vascular access, and pelvic binders all improve time-dependent patient care.

The COVID-19 pandemic overwhelmed health systems due to an enormous number of critically ill patients presenting all at once. The disease had insidiously spread far wider than governments were aware. We have learned that the associated pneumonia also advances insidiously, and by the time patients present to the ED they have moderate to severe ARDS. COVID-19 simply does not fit with our prior clinical experience with patients who have severe lung injury and hypoxia. Patients with COVID-19 pneumonia often have alarmingly low oxygen saturations (~50%–80%) but frequently do not feel short of breath. Patients who become acutely hypoxic, like those who choke or drown, rapidly become unconscious or seize. Respiratory failure patients with rapid-onset hypercarbia become narcotized and lethargic. Most patients we need to intubate in emergency have either precipitous hypoxia, hypercarbia, or shock that leads to compromised mental status. They have subjectively and clinically evident shortness of breath and dyspnea with increased work of breathing.

COVID-19 pneumonia patients often do not subjectively appreciate their lung injury. Through the virus’ effect on surfactant and resultant alveolar collapse, patients have a progressive drop in PaO₂ and an incremental increase in their respiratory rate. This process develops over days. The lungs initially remain “compliant” and patients effectively ventilate, lowering PaCO₂. They develop a large right-to-left shunt. I believe much of the lethality of COVID-19 has to do with the lack of subjective symptoms despite the advanced underlying lung injury that is occurring. It has been postulated by Gattinoni et al. that the increase in respiratory drive exacerbates the inflammation and lung injury caused by the virus itself. Gattinoni has described the intact gas volume of the lung and high shunt fraction of COVID-19 pneumonia as an atypical form of ARDS, although there remains much debate about phenotyping COVID-19 cases. Eventually, the cycle of worsening hypoxia, increasing respiratory rate, and the underlying lung injury precipitates overt respiratory failure. Acute respiratory failure, cardiac dysthymia due to severe hypoxemia, and thrombosis may explain the alarming number of COVID-19 patients found dead at home. The lack of subjective symptoms found in COVID-19 pneumonia also explains the many cases of incidentally discovered pneumonia in ED patients who present with syncope, fatigue, and other medical complaints. I wrote of this phenomenon that I called “silent hypoxia” in a New York Times opinion piece that was published on April 20, 2020.

In the two short months since COVID-19 exploded in our health care system, we have learned much about the disease. COVID-19 kills through its attack on the lungs in almost all patients. Thrombosis, renal failure, and neurologic injury largely correlate with severity of lung injury and also prolonged mechanical ventilation. The onset of pneumonia is between 5 and 10 days postinfection. Although there...
are many laboratory abnormalities involving abnormal blood counts and inflammatory markers, the single most reliable marker of critical illness (ICU care, mechanical ventilation, and death) found in a large health care system in New York City involving more than 4,100 COVID-19 cases was the level of hypoxia on presentation.5

This month’s AEM article confirms the utility of home pulse oximetry monitoring as a screening tool for COVID-19 pneumonia.6 This study validates pulse oximetry for determining the need for hospitalization. It also confirms the phenomenon of silent hypoxia, because 50% of patients who returned requiring treatment for COVID-19 pneumonia in this study had no subjective worsening of symptoms. They only returned because of close pulse oximetry monitoring.

In all areas of emergency medicine, we know that earlier detection and intervention minimizes end-organ injury and improves outcomes. I believe that this will be shown with COVID-19 pneumonia too. Last month in AEM, Caputo et al.7 reported that awake proning and positioning maneuvers coupled with non-invasive oxygenation reduced the need for intubation in two of three patients with moderate to advanced COVID-19 pneumonia. Hopefully, such techniques will work even better if we identify pneumonia earlier with only mild hypoxia and before severe lung injury. This month’s study by Shah et al.6 supports the growing body of literature that pulse oximetry monitoring should be a standard of care for discharging known or suspected COVID-19 patients.

We just crossed the 150,000 dead mark. But amidst the pessimism of this pandemic, this study of home oximetry monitoring coupled with last month’s AEM publication on proning points to progress and hope. We have learned much about how COVID-19 kills in a short period of time. It would be great if magic bullets arrive that can stop this virus instantly or vaccines appear that prevent further infections. In the short term, though, we must focus on incremental gains related to COVID-19 pneumonia: earlier detection through pulse oximetry and supportive and adjunctive care that reduces the need for mechanical ventilation.

Richard M. Levitan, MD
(airwaycam@gmail.com)
Littleton Regional Healthcare, Littleton, NH

Supervising Editor: Jeffrey A. Kline, MD

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