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Can Early Electroencephalography Findings Predict Survival and Functional Outcome in Patients With Severe COVID-19 Infection?*

KEY WORDS: acute respiratory distress syndrome; COVID-19; electroencephalography; encephalopathy; mortality

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CCOVID-19 has affected nearly half a billion people globally and is responsible for over 5 million deaths, approximately 1 million of whom are in the United States (1). Although vaccination has resulted in reduced mortality rates and severity of disease (2), COVID infections and their consequences for patients will continue. Although COVID-19 is primarily a respiratory disease, neurologic symptoms are frequent (3). Anosmia, ageusia (3), dizziness, headache, myalgias (4), cerebrovascular ischemia, and encephalitis

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have been reported (3). However, the most appropriate methods for acute neurologic monitoring for COVID-19 in critically ill patients remain unsettled.

Electroencephalography (EEG) monitoring presents challenges in patients with COVID-19. The placement of EEG leads requires close contact from technologists, modification of protocols, and potential changes in the interpretation of the study (5). Clinical seizures are typically seen in patients with history of epilepsy in the context of viral febrile illness, dehydration, hypoxemia, hypercapnia, or fever during acute COVID-19 infection (6). Critically ill patients frequently have subclinical seizures seen only on EEG, which result in delayed neurologic recovery and worse functional outcomes (7). Subclinical seizures may also occur as a complication (8). Hence, continuous video EEG monitoring is helpful (9, 10). Patients with COVID-19 often have unusual and abnormal EEG findings, such as sporadic epileptiform discharges (11), abnormal background activity, or slowing. The severity of the EEG abnormalities often reflects the severity of the illness in general (12, 13). How best to interpret EEG data in patients with COVID-19 is unclear, particularly to anticipate crucial events: awakening, delirium, and mortality.

In this issue of *Critical Care Medicine*, Benghanem et al (14) prospectively assessed EEG in patients with respiratory failure in the setting of COVID-19. EEG and structured clinical assessments were obtained within the first 12–72 hours, when paralytics were first stopped (T1) and 4–7 days after sedation was discontinued (T2) at two university hospitals. Poor reactivity of the EEG and discontinuous background in early EEGs were predictive of a longer need for mechanical ventilation, longer time comatose, more delirium, and higher mortality (41% vs 11% for poor reactivity and 40% vs 4% for discontinuity) at 28 days. The authors reasonably attempt to control for confounders, such as sedation, neuromuscular blockers, and analgesia following a standardized protocol at the institutions. It is a commendable effort for a challenging population to study. These results suggest that EEG has a promising role in anticipating complications and outcomes in patients with COVID-19, even in the setting of severe illness that can potentially confound EEG interpretation.

COVID-19 seems unlikely to be vanquished. It seems more likely to become another of the common viral causes of critical illness that leads to multisystem

illness and multiple complications. The EEG abnormalities described may be helpful to explain some of the varied consequences of COVID-19 in survivors (15). A variety of neuropsychologic symptoms (e.g., reduced cognitive function) may be ascribed to both delirium (16) and COVID-19, yet their relative contribution is unclear. The descriptions of EEG abnormalities are not specific, and future work might define abnormalities that are pathognomonic, lead to specific future events, or be recognizable by artificial intelligence algorithms. COVID-19 is becoming another endemic disease that requires neuromonitoring, anticipating complications, and prognosticating for loved ones of critically ill patients. For better or worse, COVID-19 is a part of the landscape for multiple specialties of critical care.

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Should We Intubate Pediatric Hematopoietic Cell Transplant Patients With Respiratory Failure Sooner?*

KEY WORDS: hematopoietic cell transplant; noninvasive ventilation; pediatrics; respiratory failure

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Respiratory failure in immunocompromised children is associated with mortality rates of 25–30%, with mortality exceeding 50% in hematopoietic cell transplant (HCT) patients with acute respiratory distress syndrome (1–6). The advancement of respiratory support modalities, notably high-flow nasal cannula (HFNC) and noninvasive positive-pressure ventilation (NIPPV), has transformed the care of children with respiratory failure, often resulting in a stepwise escalation across interfaces prior to endotracheal intubation and invasive mechanical ventilation (IMV) (7–9). For some patients, HFNC and NIPPV can offer adequate support without the need for sedation and invasive procedures. In contrast, IMV offers more advanced support but frequently requires sedation and carries a risk of procedural complications. Approximately 15–23% of children using HFNC or NIPPV ultimately require IMV; for HCT patients, this number may exceed 60%, likely due to a greater propensity for severe respiratory disease (5).

Therefore, it remains a complex and daily question for bedside physicians treating patients with respiratory failure to determine who should receive up-front NIPPV support and for how long before escalating to IMV. Many factors affect this decision, including illness severity, anticipated illness trajectory, comorbidities, and patient goals of care. Recent data in the pediatric HCT

*See also p. 1127.

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