

EEG Abnormalities and Their Radiographic Correlates in a COVID-19 Inpatient Cohort

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Abstract

Background and Objectives

To identify the prevalence of EEG abnormalities in patients with coronavirus disease 2019 (COVID-19) with neurologic changes, their associated neuroimaging abnormalities, and rates of mortality.

Methods

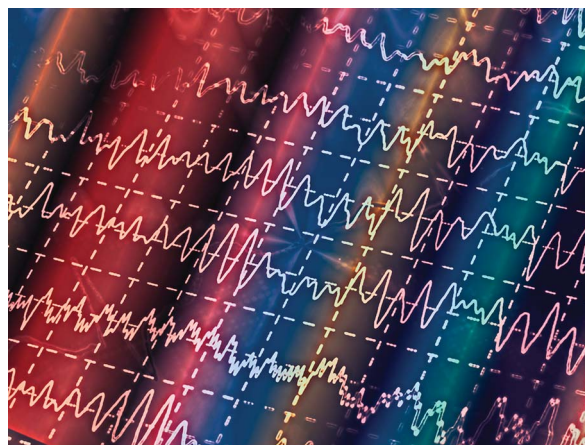
A retrospective case series of 192 adult COVID-19-positive inpatients with EEG performed between March and June 2020 at 4 hospitals: 161 undergoing continuous, 24 routine, and 7 reduced montage EEG. Study indication, epilepsy history, intubation status, administration of sedatives or antiseizure medications (ASMs), metabolic abnormalities, neuroimaging pathology associated with epileptiform abnormalities, and in-hospital mortality were analyzed.

Results

EEG indications included encephalopathy (54.7%), seizure (18.2%), coma (17.2%), focal deficit (5.2%), and abnormal movements (4.6%). Epileptiform abnormalities occurred in 39.6% of patients: focal intermittent epileptiform discharges in 25.0%, lateralized periodic discharges in 6.3%, and generalized periodic discharges in 19.3%. Seizures were recorded in 8 patients, 3 with status epilepticus. ASM administration, epilepsy history, and older age were associated with epileptiform abnormalities. Only 26.3% of patients presented with any epileptiform abnormality, 37.5% with electrographic seizures, and 25.7% patients with clinical seizures had known epilepsy. Background findings included generalized slowing (88.5%), focal slowing (15.6%), burst suppression (3.6%), attenuation (3.1%), and normal EEG (3.1%). Neuroimaging pathology was identified in 67.1% of patients with epileptiform abnormalities, over two-thirds acute. In-hospital mortality was 39.5% for patients with epileptiform abnormalities and 36.2% for those without. Risk factors for mortality were coma and ventilator support at time of EEG.

Discussion

This article highlights the range of EEG abnormalities frequently associated with acute neuroimaging abnormalities in COVID-19. Mortality rates were high, particularly for patients in coma requiring mechanical ventilation. These findings may guide the prognosis and management of patients with COVID-19 and neurologic changes.



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Neurologic manifestations of coronavirus disease 2019 (COVID-19), including altered mentation, seizures, cerebrovascular complications, encephalitis, and immune-mediated

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necrotizing encephalopathy, are well documented.¹⁻⁷ An early case series reported serious new-onset neurologic events in 3.5% of 917 COVID-19 inpatients and in 9.4% of those critically ill, with acute symptomatic seizures reported infrequently.^{1,8,9} However, in patients with COVID-19, seizures may reflect serious underlying neuropathology and portend a worse prognosis.⁷ Relying solely on observed clinical seizures, particularly when patients remain lethargic, comatose, or delirious for extended periods of time, may result in missed detection of nonconvulsive seizures.¹⁰ Therefore, the true incidence COVID-19-related seizures remains unknown, particularly concerning in critical care settings where patients face an increased risk of COVID-19-related complications including fever, respiratory failure, coagulopathy, multiorgan damage, metabolic derangement, and cytokine storming.^{7,10-13}

In addition to elucidating EEG data in the COVID-19 population, there is also a greater need to correlate related radiographic data to guide clinical care. A review of 116 patients with COVID-19 with neurologic symptoms, including seizures and status epilepticus, revealed vascular thrombosis, acute hemorrhagic necrotizing encephalopathy, encephalomyelitis, and bilateral frontotemporal hypoperfusion.¹⁴ This demonstrates the importance of imaging, with more research required to better correlate specific COVID-19-related findings to EEG abnormalities.

The New York City region was an early COVID-19 global epicenter. From March to June 2020, EEG studies were performed on 192 consecutive adult inpatients at 4 tertiary medical centers in this study. This article aims to address some of the aforementioned issues by presenting the EEG and neuro-radiographic findings along with a review of key clinical features.

Methods

A retrospective review was performed of 192 consecutive confirmed adult COVID-19–positive inpatients who underwent EEG for clinical purposes as the main inclusion criteria. All patients included were confirmed SARs-CoV-2 positive by PCR via nasopharyngeal swab and were admitted to 1 of 4 tertiary hospitals. EEGs were interpreted by a board-certified epileptologist, with continuous and routine EEG recordings using standard 19-channel recordings and disposable electrode sets (Natus, Pleasanton, CA) and an 8-channel disposable headband for reduced montage EEG (RM-EEG) (Ceribell, Mountain View, CA). Indications for EEG were categorized as encephalopathy, coma, focal neurologic deficit, abnormal movements (tremors or shivering), or witnessed seizure-like activity based on documentation. EEGs were classified as normal or abnormal; abnormal EEG findings were further subcategorized as epileptiform or nonepileptiform. The criteria for epileptiform activity included focal intermittent epileptiform discharges (IEDs), subclassified for the presence of lateralized periodic discharges (LPD); generalized periodic discharges (GPD),

subclassified as with or without triphasic morphology; and seizure, subclassified if status epilepticus occurred. Background activity was classified as normal, generalized slowing (mild, moderate, and severe), focal slowing with or without lateralized rhythmic delta (LRDA), burst suppression, and diffuse attenuation. For patients with multiday recordings, the most severe abnormalities during their hospital courses were registered in the database. For patients with epileptiform EEG abnormalities, neuroradiographic data on CT or MRI were reviewed by a board-certified neuroradiologist and analyzed and classified as either unremarkable for age, acute, chronic pertinent, or chronic and likely unrelated (such as subcortical white matter disease, lacunar infarcts, or age-related volume loss).

Descriptive statistics (median and interquartile range for continuous variables and frequency distribution for categorical variables) were calculated. Age, sex, indications for EEG, the need for mechanical ventilation, the use of continuous intravenous (CIV) sedation (benzodiazepine or propofol), antiseizure medication (ASM) administration, the presence of clinically significant metabolic abnormalities, and a history of epilepsy were screened in the univariable analysis as risk factors for epileptiform EEG or in-hospital mortality (defined as occurring within the timeframe of the study). Univariable logistic regression was performed to screen variables with a *p*-value criterion of *p* < 0.05 for entry into the model selection procedure. A stepwise selection was performed using the variable entry criterion of *p* < 0.05 and variable retention criterion of *p* < 0.05 to enter the final multivariable model. Multivariable logistic regression was performed to determine whether the selected risk factors were associated with the outcome variables.

Data Availability

All study data are available from the corresponding author upon reasonable request from any qualified researcher.

Standard Protocol Approvals, Registrations, and Patient Consents

The Northwell Health Institutional Review Board approved this case series as minimal-risk research using data collected for routine clinical practice. Northwell COVID-19 research consortium record number 147. It was determined by the IRB that written informed consent by participants was not needed because of the retrospective nature of this study and lack of identifiable personal health information being used.

Results

A total of 192 COVID-19–confirmed patients had EEGs performed from March 2020 to June 2020. The average patient age was 67 years (range 23–92), with 38% women. At least 1 day of continuous EEG monitoring was performed on 161 patients, with an average of 2.2 days of recording (range 1–12 days). Routine EEG or RM-EEG studies were performed exclusively on 24 and 7 patients, respectively.

Table 1 Patient Characteristics Correlated With the Risk of Epileptiform Discharges or Seizures on EEG and In-Hospital Mortality

Characteristic	All patients (n = 192)	Epileptiform discharges or seizures on EEG		In-hospital mortality	
		Univariate OR (95% CI)	Multivariate OR (95% CI)	Univariate OR (95% CI)	Multivariate OR (95% CI)
Median age (IQR, min, max)	70 (20, 23, 92)	1.04 (1.02–1.06) <i>p</i> < 0.001	1.06 (1.03–1.09) <i>p</i> < 0.001	1.021 (1.00–1.04)	
Females (%)	72 (38)	1.15 (0.83–2.09)		0.75 (0.41–1.38)	
EEG indications:					
Abnormal movements (%)	9 (5)	0.42 (0.09–2.08)		1.35 (0.35–5.21)	
Encephalopathy (%)	105 (55)	0.87 (0.49–1.56)		0.68 (0.38–1.22)	
Coma (%)	33 (17)	0.61 (0.27–1.37)		4.36 (1.96–9.68) <i>p</i> = 0.003	2.65 (1.14–6.17) <i>p</i> = 0.024
Focal neurological deficit (%)	10 (5)	1.56 (0.44–5.59)		0.70 (0.18–2.81)	
Seizure (%)	35 (18)	2.08 (0.99–4.37)		0.43 (0.18–1.01)	
Previous epilepsy (%)	29 (15)	4.25 (1.81–9.94) <i>p</i> < 0.001	4.59 (1.60–13.21) <i>p</i> = 0.005	0.59 (0.25–1.41)	
Mechanically ventilated (%)	106 (55)	0.71 (0.39–1.26)		4.38 (2.28–8.41) <i>p</i> < 0.001	3.42 (1.72–6.81) <i>p</i> < 0.001
CIV Sedative (%)	61 (32)	0.47 (0.24–0.91) <i>p</i> = 0.025		2.78 (1.48–5.21) <i>p</i> = 0.001	
Metabolic abnormality (%)	109 (57)	1.08 (0.60–1.94)		2.13 (1.16–3.92) <i>p</i> = 0.015	
Antiseizure medication (%)	74 (39)	2.95 (1.61–5.40) <i>p</i> < 0.001	2.48 (1.21–5.07) <i>p</i> = 0.013	0.85 (0.46–1.55)	

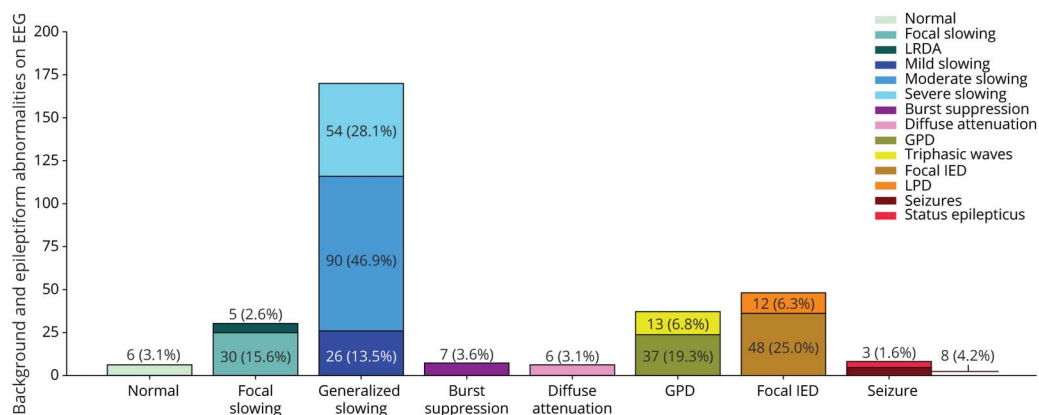
Statistically significant variables in bold. Abbreviations: IQR = interquartile range; OR = odds ratio.

Primary EEG indications were determined to be encephalopathy (54.7%), clinical seizure (18.2%), coma (17.2%), focal neurologic deficit (5.2%), and abnormal movements (4.7%) (Table 1). Only 9 of 35 (25.7%) of patients with concern for clinical seizures preceding EEG had a history of epilepsy.

Epileptiform EEG Abnormalities

Of the 192 patients, 76 (39.6%) had epileptiform discharges or concerning periodic patterns (Figure). Epilepsy history was documented in only 20 (26.3%) of these cases. Acute or pertinent chronic structural lesions were found in 51 of the 76 patients (67.1%), with 36 (47.4%) being acute in nature.

Figure Prevalence of Background and Epileptiform Abnormalities on EEG in COVID-19



Numerical values and (%) of patients among the cohort (because of patients with overlapping EEG findings, percentages do not equal 100%).

Electrographic seizures were captured in 8 patients; 3 were found to be in nonconvulsive status epilepticus (NCSE). Epilepsy history was documented in 2 patients. Imaging abnormalities were noted in all patients, with acute structural lesions in 6 patients, including ischemic infarcts, leukoencephalopathy, focal leptomeningeal enhancement, hemorrhagic cerebral brain metastasis, and recent craniotomy.

Focal IEDs were the most common epileptiform finding, seen in 48 patients (25.0%), with a previous epilepsy diagnosis reported in a third of cases. In patients without epilepsy, focal IEDs were frontal (22 cases), temporal (13 cases), parietal (2 cases), and occipital (1 case), overlapping. In patients with epilepsy, focal IEDs were frontal (7 cases), temporal (10 cases), parietal (2 cases), and occipital (1 case), overlapping. Acute or chronic structural lesions were in 35 of 48 (72.9%) patients, with 23 (47.9%) with acute lesions (Table 2). LPDs were found in 12 patients (6.3%), with a history of epilepsy in half of them. A structural lesion was observed in 9 of 12 (81.8%) patients with LPDs, 6 acute. Acute imaging abnormalities on CT or MRI included ischemic strokes with multifocal punctate lesions, cortical or brainstem lesions, watershed infarcts or hypoxic-ischemic changes; cerebral or cerebellar hemorrhagic strokes; subdural and subarachnoid hemorrhages; occipital focal leptomeningeal enhancement, gyral edema, olfactory tract inflammatory changes, T2 and FLAIR hyperintensity in the dorsomedial thalami; leukoencephalopathy; and recent tumor resection.

GPDs were found in 37 patients (19.3%), 13 (6.8%) classified with triphasic morphology (Figure). Epilepsy history was noted in 5 patients. Structural lesions on neuroimaging were found in 21 (56.8%) of these patients, 18 (48.6%) of which were acute (Table 2). Acute structural lesions included punctate multifocal infarcts, discrete cortical lesions, watershed lesions; hypoxic leukoencephalopathy; cerebellar, brainstem, or cortical hemorrhagic strokes; temporal, callosal, forniceal, mammillary body, or thalamic T2 hyperintensity; leptomeningeal enhancement; and posterior reversible encephalopathy syndrome (PRES).

An univariable analysis showed that age, CIV sedative, ASM administration, and epilepsy history were associated with epileptiform abnormalities on EEG. A stepwise model

selection retained age, ASMs, and epilepsy history in the final model. The final multivariable logistic regression model showed that age (OR = 1.06 95% confidence interval [CI]: 1.03–1.09), ASMs (OR = 2.48, 95% CI: 1.21–5.07), and epilepsy history (OR = 4.59, 95% CI: 1.60–13.21) were the risk factors for epileptiform abnormality on EEG (Table 1). Specifically for age, for every 10-year increase, the odds of having an epileptiform abnormality on EEG was 1.75 times higher than those who were 10 years younger after adjusting for ASMs and epilepsy history (OR = 1.75, 95% CI: 1.34–2.28).

Background EEG Findings

Among the cohort, 88.5% of patients demonstrated generalized slowing (13.5% mild, 46.9% moderate, 28.1% severe), 3.1% burst suppression, 2.6% diffuse attenuation, and 3.1% normal recordings. Thirty patients (15.6%) had focal slowing, 5 characterized as LRDA (Figure).

Mortality

Coma, use of mechanical ventilation, CIV sedative, and metabolic abnormalities were shown to be associated with in-hospital mortality in an univariable analysis. The stepwise model selection retained coma and the use of mechanical ventilation in the final model. The final multivariable logistic regression model showed that coma (OR = 2.65, 95% CI: 1.14–6.17) and the use of mechanical ventilation (OR = 3.42, 95% CI: 1.72–6.81) were the major risk factors for in-hospital mortality (Table 1).

The overall in-hospital mortality rate was 37.5%. Of 76 patients with epileptiform abnormalities, 30 (39.5%) expired, compared with 42 (36.2%) patients without epileptiform abnormalities. The rates of mortality were highest in patients with GPDs, which was 43.2%, up to 53.8% in patients with triphasic GPDs, followed by patients with focal IEDs (39.5%), although up to 41.6% in cases with LPDs. Only 1 of 8 patients with electrographic seizures expired (Table 2).

Discussion

This study represents the largest COVID-19 EEG database in the literature, with a high proportion of patients demonstrating epileptiform findings and nonreassuring periodic

Table 2 Association of Specific Epileptiform Abnormalities on EEG With a History of Epilepsy, Neuroimaging Abnormalities, and In-Hospital Mortality

Epileptiform abnormalities (n)	Epilepsy history	Acute or chronic structural lesion on neuroimaging	Acute structural lesion on neuroimaging	In-hospital mortality
Focal IEDs (48)	33.3% (16)	72.9% (35)	47.9% (23)	39.5% (19)
GPDs (37)	13.5% (5)	56.8% (21)	48.6% (18)	43.2% (16)
Seizures (8)	37.5% (3)	75.0% (6)	75.0% (6)	12.5% (1)
Any epileptiform abnormality (76)	26.3% (20)	67.1% (51)	47.4% (36)	39.5% (30)

patterns.¹⁵⁻¹⁸ The in-hospital mortality of this cohort of COVID-19 inpatients requiring EEG was relatively high (37.5%), although consistent with the mortality rate of 44% found in another New York-based hospital system around this period.¹⁹ A considerable number (18.2%) of patients in our study had documented events of clinical concern for seizure before the EEGs being ordered, and a very high proportion of patients presented with encephalopathy or coma. Over a third of patients in our cohort demonstrated overt epileptiform spiking or periodic patterns of concern, although seizures were relatively infrequent, observed in 8 patients (4.3%). For comparison, 1 previous study noted a smaller percentage with epileptiform abnormalities (27%) and a higher number of patients with recorded seizures (7%).¹⁹ However, within our cohort, 3 patients were found to be in NCSE, a particularly serious and treatable condition. Older age, and not surprisingly, ASM administration and epilepsy history were significantly associated with epileptiform abnormalities on EEG. However, only approximately a quarter of the patients with epileptiform abnormalities or electrographic seizures on EEG had known epilepsy, suggesting that most findings were *de novo*.

In many reports of COVID-19, EEG data are conspicuously lacking. This multifaceted problem may reflect limited availability of EEG and subspecialists in nontertiary hospitals or a purposeful reduction in EEG procedures to decrease exposure risk. Most patients in this cohort had at least 1 continuous EEG recording, with extensive electrographic data and greater chance of recording pertinent abnormalities. The relatively novel approach of RM-EEG was also used, technology potentially suited to the critical care setting for rapid evaluation for seizures. The potential therapeutic implications of EEG studies must be weighed carefully against increased SARS-CoV-2 transmission risk to both patients and healthcare staff. Recent recommendations include utilization of personal protective equipment, disposable electrodes, RM-EEG, and avoiding hyperventilation as an activating procedure.²⁰ In our study, precautions included the above protocols and clinical prescreening, and reduced staffing models. In a previous study of 26 critically ill patients with COVID-19 with altered mentation, most showed diffuse slowing on 30 minute EEG recordings; however, 2 patients had isoelectric EEG consistent with brain death and 5 cases demonstrated frontal maximal GPDs similar to those in this report.¹⁵ Three of the 5 cases of GPDs were associated with myoclonus, and 3 of those patients expired. At least 1 patient had this pattern in the setting of acute encephalopathy without intubation or sedation and diffuse white matter hyperintensity on MRI with normal lumbar puncture results.

A study from France of 36 patients with COVID-19, half critically ill, also showed GPDs or multifocal periodic discharges in 32.5% of routine EEGs, 3 performed in patients with unexplained encephalopathy.¹⁶ EEG was normal or only mildly abnormal in about half of the cases.

Galanopoulou et al.¹⁷ studied 22 COVID-19–positive patients with 2 routine EEGs and 20 RM-EEGs. Sporadic epileptiform discharges were recorded in 40.9%, with frontal maximal sharp waves present in 8 of 9 patients. Periodic or rhythmic discharges occurred in 18.2% of cases. EEG characterization was limited by the predominant use of RM-EEG used to a greater proportion compared with this study.

In an Italian cohort of 15 patients with COVID-19–related encephalopathy, all but 2 patients with anoxia had either severe attenuation or status epilepticus, all with varying degrees of slowing, although without periodic activity noted.¹⁸ One patient demonstrated frontal intermittent generalized rhythmic delta activity. Elevated CSF protein was demonstrated in 1 patient, and MRI showed T2 hyperintensities in 2 patients.

Researchers have pondered whether GPDs may be an EEG pattern specific to COVID-19 encephalopathy.²¹ It is more probable that GPDs reflect severity of illness as opposed to a pathognomonic finding because GPDs are common, in up to 4% of patients in critical care settings undergoing EEG monitoring. Associated etiologies typically involving diffuse cortical or thalamocortical dysfunction.²² GPDs may increase the risk for associated electrographic seizures in at least 16% of patients.²³ Features such as motor manifestations, faster discharge rate, and response to ASMs may be associated with a higher risk of GPDs evolving to seizures or representing NCSE. Follow-up EEG monitoring, trials of nonseizure ASMs, or short-acting benzodiazepines may be warranted to assess clinical response.

The significance of GPDs with triphasic morphology is more controversial and classically described, associated with metabolic encephalopathy. Recent literature supports their occurrence with multiple underlying etiologies—only modest interrater EEG reader agreement regarding triphasic classification, but increased risk of seizures similar to GPDs without triphasic morphology.²²

Focal IEDs, LPDs, or LRDA were observed in a quarter of patients in our study, indicating an elevated risk of focal onset seizures. A recent Critical Care EEG Monitoring Research Consortium study showed LRDA and LPDs, in particular, associated with seizures in 28% and 44% of EEG studies, respectively.²³ Seizure risk approached nearly two-thirds of patients in highest risk subgroups, with a high frequency of discharges and intermixed fast frequency activity or spiking.

In this study, we correlated epileptiform EEG findings with radiographic abnormalities to understand what proportion of patients with these EEG findings may be secondary to new acute lesions related to COVID-19 or because of exacerbation of preexistent lesions. As stated, focal IEDs were found in a quarter of patients, LPDs in 6.3%, and focal slowing in 15.6% of cases, hence raising concern for focal cerebral pathology. The presence of focal epileptiform or periodic EEG

abnormalities was associated with a high rate of neuroimaging findings (72.9%), with acute lesions identified among two-thirds of those. Acute imaging abnormalities included ischemic and hemorrhagic stroke, watershed lesions, hypoxic leukoencephalopathy, subdural and subarachnoid hemorrhages, focal inflammatory changes, leptomeningeal enhancement, and PRES. Likewise, GPDs were found in a relatively high proportion of cases (19.3%), likely reflecting severe patient condition, depth of encephalopathy, and more widespread or global cerebral dysfunction because of metabolic factors, anoxia, infection, or inflammatory factors in critically ill patients with COVID-19. Indeed, in this study, patients with coma were 2.6 times more likely to expire during the hospital stay after adjusting for mechanical ventilation use.

The data presented in this study reflect a range of imaging neurologic abnormalities potentially associated with COVID-19. Although it is difficult to determine causality in our cohort, some findings may reflect a prothrombotic state because of immune activation, endothelial cell injury secondary to ACE2-mediated viral tropism, hypoxic ischemia, or cytokine storming. These pathophysiologic mechanisms may lead to acute infarcts with heavy clot burdens, intracranial hemorrhages, leukoencephalopathy, global hypoxic injury, cytotoxic lesions of the corpus callosum, olfactory bulb involvement, and cranial nerve enhancement.²⁴

In a large retrospective study from China, new-onset serious neurologic complications were noted in 32 of 917 patients, mostly stroke or altered mentation.⁹ The authors concluded that evidence for CNS involvement because of SARS-CoV-2 was lacking; however, in attempting to reduce virus transmission, no patients were evaluated by MRI or EEG. Head CT was performed in only 9 cases. However, several subsequent case reports demonstrated CNS-related complications including acute necrotizing encephalopathy, demyelination, stroke, cortical venous sinus thrombosis, PRES, and seizures, indicating serious potential neurologic effects of SARS-CoV-2, possibly from direct CNS invasion or secondary effects from autoimmunity or hypercoagulability.^{7,25,26}

In a recent review of 61 studies published between 2019 and 2020, radiographic abnormalities were differentiated on mild or severe clinical presentation secondary to COVID-19. Radiographic abnormalities most often included white matter abnormalities, ischemic stroke, and intracerebral hemorrhage.²⁴ Less often, abnormalities included leptomeningeal enhancement, venous thrombosis, PRES, olfactory bulb lesions, and encephalitis.²⁴ The diverse neuroradiographic findings and overlap of severity indicates the utility of radiographic imaging for the management and treatment patients with COVID-19.

Limitations of this study include the retrospective nature of the analysis because causal relationships between the variables presented cannot be drawn from the data. There was

inherent variation in clinical decision-making between providers during COVID-19 redeployment, including when an EEG was ordered and, at times, in the absence of neurologic consultation. The high mortality rate may have reflected triaging of EEG resources and a selection bias toward recording more neurologically ill patients, where EEGs less likely to affect clinical decision-making were not performed because of concerns for contagion. Because our data were derived from a large healthcare system with 23 regional hospitals, patients transferred for higher levels of care to the tertiary hospitals may have created a referral bias toward more severe cases. This cohort represents a fraction of the estimated >15,000 patients with COVID-19 admitted to the health system during this time, specific to inpatient COVID-19 cases with neurologic complaints and not necessarily relevant to lesser acute cases. The duration of this study was limited and thus did not provide long-term outlook for persistent electrographic abnormalities or neurologic outcome. Additional variables such as the presence of superinfections, CSF, and cytokine laboratory values may be subject to further study.

This study represents the largest series of EEGs in patients with COVID-19 and highlights the importance of fully assessing neurologic complications that may occur, especially in the inpatient or critical care setting. Epileptiform and periodic patterns on EEG were frequently observed and often associated with the presence of acute neuroimaging abnormalities. Mortality rates in this cohort were high and most elevated in patients with periodic EEG patterns such as GPDs and LPDs, with coma and mechanical ventilation requirements as major risk factors. EEG in patients with COVID-19 may play a critical role in detection of seizure-related patterns, identification of patients at greater risk for neuropathology, and prognostication. Patients with focal or potentially epileptiform patterns raise concern for acute brain lesions and may warrant neuroimaging evaluation. Specific patterns may signal need for further interventions such as ASMs, stroke therapies, or immunomodulatory therapy. Severe EEG abnormalities such as diffuse attenuation, burst suppression, and GPDs may be concerning for anoxia and the possibility of poorer prognosis. Although the data support seizures as a rare occurrence, their presence is concerning for neurologic complications associated with SARS-CoV-2. Most patients with electrographic and observed clinical seizures before EEG in this study had no known history of epilepsy.

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Noah Markowitz, BS	Feinstein Institutes for Medical Research, Manhasset, NY	Major role in the acquisition of data and analysis or interpretation of data

Appendix (continued)

Name	Location	Contribution
Tung Ming Leung, PhD, MPH	Feinstein Institutes for Medical Research, Manhasset, NY	Analysis or interpretation of data
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